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SECOND WORLD CONGRESS OF CARDIOLOGY AND TWENTY-SEVENTH ANNUAL SCIENTIFIC SESSIONS OF THE AMERICAN HEART ASSOCIATION

Held in Washington, D C.

WORLD TRENDS IN CARDIOLOGY

I. Cardiovascular Epidemiology
II Cardiovascular Surgery
III Blood Volume and Contractile Protein in Heart Muscle
IV. Cardiovascular Diagnosis and Therapy

World Trends in Cardiology: V

Instrumental Methods in Cardiac Diagnosis

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A HOEBER-HARPER BOOK

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INSTRUMENTAL METHODS IN CARDIAC DIAGNOSIS

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FOREWORD

This volume brings together those sessions of the Second World Congress of Cardiology which dealt with instrumentation Its contents illustrate several of the types of sessions held at that Congress Part I deals with diagnostic instrumentation, particularly as used in congenital heart disease, and it is proper that the chairman of this symposium was Dr Andre Cournand who did so much to develop the application of catheterization for clinical diagnosis The formal presentations of the several distinguished participants show how much instrumentation developed between 1950 and 1954

Part II contains a presentation of memorials to two of the masters in electrocardiography: Dr Frank Norman Wilson, whose death between the two World Congresses was widely mourned, and Dr Willem Einthoven who, fifty years before this Congress, described the string galvanometer which bears his name.

Part III contains two panels, one on electrocardiography, the other on ballistocardiography—the former an established clinical procedure, the latter still seeking its place in clinical practice

The panel on electrocardiography was unique in international congresses, since a distinguished panel of experts from Europe and the Americas interpreted as *unknowns* electrical records of patients who later died and were autopsied, so as to test their ability to diagnose the state of these diseased hearts The ordinary technique of electrocardiography was placed in competition with stereovectorcardiography, so as to compare

the relative merits of the two types of recording. Not only was this an interesting exercise in evaluating the practical utility of electrocardiography, but it demonstrated that a controversy can be aired as effectively by a panel of experts from different countries around the world as it has come to be expected when a panel is confined to residents of one country.

The panel of ballistocardiography was of a different type. It presented a broad perspective of the status of this method in 1954, ranging from its mathematical and physical background to its scientific and clinical utility. Some interplay among the distinguished participants and some experts in the audience rounded out a forthright presentation of current opinion, in which Dr. Isaac Starr, in many ways the modern founder of ballistocardiography, participated not only as chairman and moderator but as an active discussant.

In editing this volume, we have attempted to keep its contents brief and the language as informal as that employed in the oral presentations.

It is with regret that the presentation "The Visual Demonstration of Congenital Malformations of the Heart by Means of the Trajectory and Position of the Catheter" by Dr. Rodolfo Limón, of Mexico City, is omitted from this publication because of editorial limitations.

L. N. K.
A. S. C.

CONTENTS

PART I

Diagnostic Instrumentation

Chairman:
ANDRE COURNAND, New York, New York

1

Diagnostic Instrumentation in Congenital Heart Lesions with Reduced Pulmonary Circulation

MAURICE CAMPBELL, London, England

It is a great honor to open this discussion on cardiac catheterization and angiocardiography in the diagnosis of congenital heart disease. I have no great technical knowledge of these procedures, but that is irrelevant because the subject is their value in ordinary clinical work, and it is from that viewpoint that I shall discuss them I would not like it thought that I do not appreciate the enormous importance of both of these methods in advancing our knowledge of the hemodynamics of the circulation and making it possible to diagnose, on clinical grounds alone, many congenital abnormalities. If I say that I do not think a method is of much importance I ask you to remember *this qualification, namely, the immense value that the* method has been to all of us in adding to our knowledge

All of you may remember your first love, but most of you found someone more attractive in later, mature judgment, someone you finally married. Angiocardiography is a first love, fascinating and beautiful, but without that permanent appeal that leads to a lasting place in one's affection. Catheterization is

more like the permament union. If I may use an American expression angiocardiography is the "first date," and catheterization, the girl you are finally "hitched to."

You may think this is unfair to angiocardiography, but I feel that there is a basis of truth in it, because angiocardiography generally fails to prove the diagnosis conclusively. It provides wonderful illustrations of what is happening and, with clinical details, it may often give one a very strong certainty of what the condition is, but it does not provide the same certainty about the findings that one gets in many cases from cardiac catheterization

I would like to put one general opinion before you. these methods do not make a diagnosis of the whole condition. They diagnose certain physiological and functional effects which must then be pieced together They diagnose a right-to-left shunt through an atrial septal defect, or a right-to-left shunt into the overriding aorta, or some obstruction to the pulmonary blood flow. Having obtained this invaluable physiologic data from special investigations, this data must then be fitted into the rest of the picture to make a complete diagnosis.

It is from the point of view of these separate entities, the right-to-left shunt and the pulmonary obstruction, that I wish to correlate the remarks I am making, rather than from the point of view of individual diagnoses

First of all, a few words about catheterization. A few things can be learned from the position of the catheter, but it is never desirable to draw too many conclusions from that alone without confirmation based on corresponding oxygen content and pressure values. But it does show one thing—the path through the patent ductus arteriosus which often gives an absolutely certain diagnosis.

Also, facts can be collected following operative treatment in Fallot's tetralogy. I have such data showing a great increase in pulmonary flow in Fallot's tetralogy after successful surgery. It rose from a preoperative average of 47 per cent to a postoperative average of 115 per cent of the systemic flow and the corresponding rise for arterial oxygen saturation was from 73 to

91 per cent This represents an average of a large number of cases The increased pulmonary flow and the increased oxygen saturation is produced without any increase in the pulmonary arterial pressure I think these figures give information obtainable from catheterization, which cannot be obtained in any other way.

Catheterization results in a large number of cases of pulmonary stenosis with closed ventricular septum show that the pressure in the right ventricle is reduced to half, without any change in the pulmonary arterial pressure In acyanotic cases, we find no change in the arterial oxygen In the cyanotic cases, with the shunt through the atrial septum, there is a 37 to 70 per cent *reduction in the amount shunted. I think this shows the detailed* information that one can get only from catheterization.

Coming to angiocardiography, I first want to take up the right-to-left shunt That, I think, we have demonstrated in every case with one exception, and I don't know why we failed there on two occasions Actually one should be certain of the right-to-left shunt without angiocardiography. But angiocardiography can generally give one the site of the right-to-left shunt

We can also demonstrate the simultaneous filling of the aorta and pulmonary trunk that is characteristic of the main bulk of right-to-left shunts, including the right-to-left interatrial shunt which, while often well shown, is more difficult to demonstrate because it is a shunt of a smaller proportion One has to look very carefully for these small interatrial shunts, because only a portion of the blood is passing through them But they can be demonstrated

Also, a ventricular septal defect can be distinguished from overriding aorta by using the multiple method, with its more exact timing You may think that the uncertainty of diagnosis *from angiocardiography can be entirely overcome by the multiple* frame methods and electrocardiographic timing, but I do not believe that this will remove all the uncertainty.

Turning to the question of obstruction in the pulmonary circulation, I think it is difficult to demonstrate where the stenosis is I also think that one cannot say *stenosis is excluded when the*

pulmonary arteries fill fairly quickly, because they do fill quite quickly in severe cases of stenosis. It is better to consider the lung filling as a whole rather than to use any specific time of filling.

Most workers are agreed that in only a proportion of their cases can they settle whether the stenosis is valvular or infundibular. At one time, when Sir Russell Brock was interested in the direct operations and we were trying to make this diagnosis preoperatively in all cases, we thought this was a very important field of angiocardiography, but as things have developed, we have come to think that catheterization during the operation is the better and more reliable way of deciding the site of the stenosis. It is vital that a double stenosis should not be missed in these cases, and even where one recognizes a valvular stenosis, one must always be on the lookout for an infundibular one as well.

In tricuspid atresia, angiocardiography provides more useful information, as a rule, than catheterization, which provides very little, because failure to enter the right ventricle may be due either to tricuspid atresia or to a technical difficulty. One does not have a feeling of certainty with a diagnosis of tricuspid atresia based on catheterization.

I feel that angiocardiography has very little place in simple pulmonary stenosis with a closed ventricular septum. It often shows a dilated pulmonary trunk beyond the stenosis, and this remains full; but, again, these are suggestive points rather than proof. It seems to me that catheterization is the important procedure here, not so much for diagnosis, but to measure its degree. The pressure gradients across the pulmonary valve are often so surprisingly high, judged by what one would expect on the basis of symptoms, that I think cardiac catheterization has great practical value, and it is our custom to use it for all cases.

This covers the commoner groups with diminished blood flow to the lung, which can be recognized easily. When one turns to a difficult case—and by that I mean when a diagnosis cannot be made after the first outpatient session, radioscopy and electrocardiography—then I think both procedures are always

needed because both are so liable to reveal something not obtained by the other. I would, therefore, urge that where there is doubt about the diagnosis, both procedures be used

The last group that I want to speak about is pulmonary atresia, in which I consider angiocardiography valuable because it is a very difficult problem to decide whether the arteries beyond the atresia are going to prove adequate for any surgical treatment. Direct operations are nearly always out of the question because the atresia is more than a closed valve, it is a longish area that is obstructed and, therefore, in considering an anastomosis one is particularly anxious to know on angiocardiography what one can visualize.

In brief, one may find abnormal collateral vessels that are larger than the pulmonary ones, in which case one is unlikely to be able to help the patient surgically On other occasions, one may find a pulmonary artery that seems to be adequate for surgical treatment. But even here, direct vision radioscopy plays quite an important part and sometimes has to be fallen back on, even after angiocardiographic investigation.

In closing, I wish to say that in spite of my remarks in favor of catheterization, I have spent much more time talking about angiocardiography. That, I think, is only a further illustration of its charm and attraction. It can produce such beautiful pictures that one is tempted to spend all one's time on it. Actually one does not get the same solid basis of information in most conditions that is obtained from catheterization.

2

Diagnostic Instrumentation in Congenital Heart Lesions with Increased Pulmonary Circulation

PIERRE SOULIÉ, Paris, France

In congenital heart disease, with an increase in pulmonary flow, interest in catheterization of the heart is three-fold.

a. to make a diagnosis;
b. to study the physiopathology;
c. to determine the indications for surgery.

As Dr. Campbell, who has favored catheterization over angiocardiography in the study of congenital heart diseases, has said, catheterization does not give a final diagnosis or an answer as to whether surgery is indicated. We have studied the arteriovenous shunts of the heart, including the interventricular communications, interauricular communications, and the Eisenmenger's complex, anomalous venous drainage; transpositions; patent ducti and aorta-pulmonary fistulae. But we shall discuss here only interauricular and interventricular communications.

What is the value of catheterization, its absolute and its relative value? An examination of the catheter on the screen as it is passed through the heart is valuable when confirmed by the oxygen content at the severed locations. The evidence of a shunt,

while useful, cannot be put in quantitative terms There exist, as a matter of fact, causes of error in the blood gas analyses. They are known, *and they have as their cause the lack of homogeneity* in the blood of different cavities. The absence of homogeneity is greatest in the presence of a shunt, but if laminar flow is frequent in the left circulation, it is less so in the right circulation In left-to-right shunts the highest values of oxygen are at the level of the communication.

Two causes of error are possible (1) when catheterization shows an increase in oxygen as you progress from the vena cava, (2) when thrombosis occurs—a possibility which is found in less than 10 per cent of the cases. When there is a progressive increase in oxygen, the location of the communication is not accurately determined Finally, the cases having two sites at which shunting occurs, for example, an interventricular communication with a ductus, offer real diagnostic problems

In case of a patent ductus arteriosus in its classic form with a machinery murmur, the diagnosis is certain and catheterization *only confirms it But catheterization is indispensable in order to* identify ducti associated with hypertension and cyanosis

Of 85 ducti catheterized by my group, the catheter was passed through the ductus in 68 per cent of the cases There was a significant blood oxygen difference between the right ventricle and pulmonary artery in 94 per cent of the cases The volume of the shunt varied from 20 to 60 per cent of the output of the left ventricle The pressure in the pulmonary artery was normal in 41 cases, hypertension was present in 44 cases, with systolic pressure less than 60 mm of mercury in 28, and between 60 and 120 mm in 16 cases. In severe cases, the pulmonary artery pressure may be higher than in the aorta

Catheterization of the heart is especially interesting in the atypical ducti with pulmonary hypertension, because it is in these cases that one finds only a systolic murmur and pulmonary congestion, which might suggest either an Eisenmenger's complex or intraventricular communications with aortic insufficiency.

Now, I turn to a second group of cases, those with an auricular

communication. These lesions are usually characterized by a murmur in the second and third intercostal spaces, marked by dilated pulmonary arterial system and a right bundle branch block. But the murmur may be atypical, the pulmonary artery only moderately dilated and the bundle branch block may be absent, the last is seen in only two-thirds of the cases. Therefore, one must understand the value of catheterization.

The catheter, in 70 per cent of the cases, passes through the interauricular communications and the oxygen analysis is significant, with an oxygen difference of more than one and a half volumes in 72 per cent of the cases Even with catheterization, one-fourth of the cases are in doubt because they fail to show a typical oxygen rise

Finally, catheterization might be misleading when the increase in O_2 content only appears at the ventricular level, making one believe that an interventricular communication is present. This happened in three of our patients, and one catheterized three years later showed a marked increase in O_2 content at the atrial level.

Catheterization evaluates the physiopathologic state. The volume of the shunt is variable. More than half of the cases show a small shunt, less than 30 per cent of the pulmonary flow. In the rest, the shunts vary from 35 to 80 per cent.

Pressures are normal in the pulmonary arteries in most of the cases and are high, from 60 to 80 mm. of mercury, in about 10 per cent.

As for interventricular communications, the diagnosis depends on the midsystolic murmur and thrill in the lower third of the sternum. But there are cases with a typical murmur, without any shunt, and shunts with atypical systolic murmurs. Catheterization is valuable when the signs are negative or atypical clinically.

Blood gas differences in interventricular shunts are usual and striking, the increase being from 2 to 3 volumes per cent. The blood is arterialized in three-quarters of the cases, and partially so in the remainder.

While we have noted the absence of shunt in 24 per cent of

interauricular and 6 per cent of the ducti, this absence of shunting is rare in the interventricular communication This is due to the large difference in pressure between the two ventricles. Passage of the catheter through the interventricular communication is rare. We have gotten it through only on two occasions. The risk of overlooking an interventricular shunt is slight

Shunts between ventricles vary from 25 to 45 per cent of the pulmonary flow and sometimes reach 70 per cent. The pressure in the pulmonary arteries may be normal, or within the limits of normal, in Roger's disease, but this is rare in an interventricular communication with a large pulmonary artery. Of 18 cases, 14 had pressures between 40 and 80 mm of mercury, and sometimes there is equality of pressure in the two circulations.

Of all the deformities discussed, associated with an increase in pulmonary circulation, interventricular communication is the one which most favors the development of increased pulmonary vascular resistance. What is the value of catheterization in the diagnosis of interventricular communication? There are three aspects:

(1) Catheterization alone, with some reservations, is able to identify deformities such as an interventricular septal defect with a large pulmonary artery with diastolic murmur or with atypical systolic murmurs.

(2) We have been able to find moderate pulmonary stenosis and see that these stenoses are associated with dilatation and expansion of the pulmonary artery. Thus, a new chapter has been opened on dilatation of the pulmonary artery and pulmonary stenosis.

(3) Drops in pressure may be seen between the right ventricle and pulmonary artery in cases of interventricular communication with large pulmonary artery, but this constriction may be functional, as in the first operative cases recently published by Blount, but a fall of more than 20 mm of mercury always makes us suspect organic stenosis.

Finally, catheterization is vital in differentiating interventricular communication with a large Eisenmenger complex. Clinically speaking, the electrocardiogram and x-ray do not permit

us to distinguish between the two conditions, and cyanosis, which has great diagnostic value, is not a perfectly reliable sign Catheterization and angiocardiography may be decisive.

In interventricular communication, where the oxygen content of the arterial blood is normal and the pressure is normal or increased, the pulmonary vascular resistance is elevated (in about one-half of the cases) but is less than the systemic peripheral resistance

In the Eisenmenger complex the arterial oxygen saturation is under 90 per cent The pressure in the pulmonary artery is equal to that of the aorta. The resistance in the pulmonary tree equals that of the systemic. Opacification of the aorta is early. Thus the diagnosis rests on the position of the aorta which is normal in interventricular septal defect and biventricular in the Eisenmenger complex.

The problem is difficult in the Eisenmenger complex since there are a variety of hemodynamic disorders associated with it. In a small number of cases, we have seen lower pulmonary resistances and bidirectional shunts. There are cases of the Eisenmenger complex which are not cyanotic, occurring in people between 15 to 20 years of age In them the picture is that of an isolated interventricular communication. The passage of the catheter in the aorta is the only way possible of avoiding this error

All this leads me to the conclusion that the problems are real. We cardiologists are already considering—and we hope it may occur in the very near future—that the surgical obliteration of interventricular communications will be done with the same ease as the interauricular procedures are now being performed.

3

Angiocinematography

F. GROSSE-BROCKHOFF, Dusseldorf, Germany

Angiocinematography, already developed by Dr. Janker in 1930, has been employed as a diagnostic device in cases of congenital heart disease by Janker and Schaede. Up to now, we have used this method in more than 650 angiocardiographs Before explaining the practical use of this method, I want to point out that the fluorescent screen cinematography is only one of the methods being used by us. Depending on the apparent conditions, the following methods are employed:

First, mechanisms of different design for direct serial exposures of from one to 6½ snaps per second.

Second, indirect serial exposures from the fluorescent screen on a 70 mm. film with a speed up to 6 snaps per second

Third, fluorescent screen cinematography with 35 mm film, giving a speed up to 24 snaps per second.

Fourth, fluorescent screen cinematography with 70 mm. film, giving the same speed of snaps as the third

Fifth, serial snaps and cinematography by means of screen amplifier have been started several months ago

All these methods have been developed and constructed in the institute of Professor Janker, and all investigations were carried out there, too

In principle, the choice of the respective method depends

upon whether a maximum number of snaps are wanted, or whether, as far as possible, detailed registration of the contrast filling is desired. The indications for the use of each of these methods will be defined. First, I want to talk about the use of cineangiocardiography. I will restrict myself to the demonstration of the fluorescent screen cinematography with 35 and 70 mm. film, which is used by us for the most part.

I will describe the arrangement of our apparatus The x-ray room is separated from the switchboard by a lead-lined wooden wall Immediately next to this wall is the table, which is protected on all sides by lead and also against light. During the last year, this arrangement was modified as follows: Now the table can be positioned as to make possible examinations in the recumbent, semi-recumbent, and upright positions.

The picture of the fluorescent screen is reflected by a mirror and taken by a Leitz lens of 1.0.0 85 light intensity of the Ascania camera of Janker The x-ray apparatus used in cineangiocardiography can deliver 120 kv. There is a device for diminishing the load of the skin which allows intermittent switching of the valve up to 24 times per second. The fluorescent screen can be observed from the switchboard during the taking of the snaps The aperture is designed in such a way that the observer is also able to watch the fluorescent screen during catheterization. To protect the staff from x-rays, the patient is completely encircled by lead-protected wooden plates, leaving free only the arm for the injection of the contrast medium.

I want to make one remark on the question of radiation x-ray and the protection against it Circumstantial and detailed statements about this, as well as all methods and the evaluation of the results of the different methods, are given in the recent monograph of R. Janker. In brief, I can say that despite the extremely great number of exposures, the load of radiation in cineangiocardiography is not essentially higher than with direct exposures using serial apertures. Irradiation of the skin is in the permissible range if proper care is taken.

The following advantages of angiocardiocinematography may be pointed out:

First, the large number of exposures—18 to 24 per second—makes it possible to study many more phases of the contrast-filled heart than is possible with any other method. Each exposure of the film can be enlarged and evaluated.

Second, all the exposures can be viewed in rapid succession in motion picture fashion. This permits recognition of hemodynamic peculiarities not detected by random single exposure. These are excellent for teaching purposes.

Third, the use of a 35 mm film is incomparably cheaper than the use of a 30 cm film

Fourth, the combination of cinematography with serial snaps, in the second plane, proves to be especially profitable By this means, the very fine demonstration of detail in the film is balanced by the simultaneous direct serial snaps in the second plane.

4

Indicator Substances in the Diagnosis of Congenital Heart Disease

HOWARD B. BURCHELL, Rochester, Minnesota

Dr. Cournand and members of the Congress: In our experience, the injection of indicator substances into the circulation and the recording of the dilution pattern at a distant site have been of immeasurable value in the accurate diagnosis of congenital heart disease.

In our laboratory, the indicator substance has been Evans blue dye, and the site of injection has been some part of the heart or great vessels, and the time of its appearance and subsequent concentrations in the arterial stream are recorded by the oximeters and technics developed by Dr Earl H Wood.

The normal dye-dilution curve from the radial artery after intravenous injection can be plotted readily The oximeter registers the increased optical density caused by the dye, and thus an increasing dye concentration is represented by a downward deflection. The normal curve shows a normal appearance time, rapid build-up of the concentration, and rapid disappearance slope. The normal, small recirculation peak is seen about 20 seconds after the maximal concentration peak (Fig 1).

Characteristic curves from the intravenous injection of dye in the peripheral circulation are seen in both types of intracardiac

shunts, that is, left-to-right and right-to-left. In the characteristic left-to-right shunt, such as occurs in patent ductus arteriosus, ventricular septal defect and atrial septal defect, the disappearance slope is distorted because of prolonged pulmonary recirculation of the dye, and the ratio of the disappearance time to the build-up time may roughly quantitate the shunt.

When a right-to-left intracardiac shunt is present, the curve

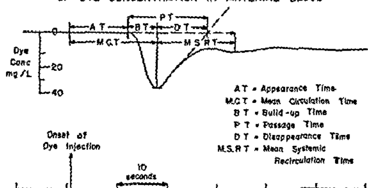


FIG. 1

differs markedly from the normal (Fig 2) in that there is an early appearance of the dye and initial peak concentration, related to direct shunting into the aorta, and a second concentration hump follows, related to that portion of the dye passing through the more nearly normal pulmonary pathway. From the ratio of the calculated areas subtended by the two humps, one may obtain a reasonably accurate calculation of the amount of the shunt.

The dye curves which follow the intravenous injection of dye thus give one an excellent screening test for the presence or absence of an intracardiac shunt, but do not localize the shunt.

When the dye-injection method is combined with cardiac catheterization, much more nearly exact information concern-

4

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Characteristic curves from the intravenous injection of dye in the peripheral circulation are seen in both types of intracardiac

(3) The demonstration of frequent preferential drainage of blood from the right lung to the right atrium in the presence of atrial septal defect.

(4) The diagnosis of the complete abnormal drainage of blood from a single lung into the right atrium or its tributaries.

(5) The demonstration of the preferential shunting of blood coming from the inferior vena cava across an interatrial communication, as compared with blood coming from the superior vena cava.

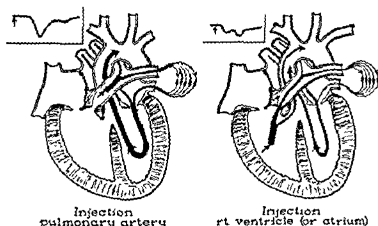


FIG 3

(6) The illustration that venous-arterial shunts in some patients may vary inversely according to the oxygen tension of the inspired air.

As the first example, I wish to portray how a right-to-left shunt may be localized at the atrial or ventricular level

Figure 3 is a diagram of the heart of a cyanosed person with pulmonary stenosis and a ventricular septal defect If the injection is made into the heart through a cardiac catheter, distal to the site of the shunt, that is, into the pulmonary artery, a normal dye curve is obtained, whereas, if the injection is made proximal

to the shunt, into the right ventricle, the dye appears early in the aorta and the characteristic double-hump dilution curve of the right-to-left shunt is obtained. If the shunt had been at the atrial level, the dye curve after injection into the right ventricle would have been normal, and that after injection into the atrium, abnormal.

In many patients in whom the classic clinical and laboratory findings of atrial septal defect are made, in addition to the large left-to-right shunt, small transatrial right-to-left shunts have been demonstrated This shunt may arise from a small portion of

DEMONSTRATION OF
PREFERENTIAL RIGHT TO LEFT TRANS-ATRIAL FLOW FROM
INFERIOR AS OPPOSED TO SUPERIOR VENA CAVA
(2 CASES OF ATRIAL SEPTAL DEFECT)

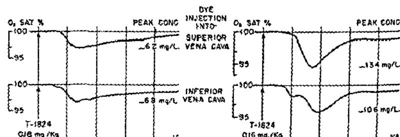


Fig. 4

superior vena caval blood crossing to the left atrium, but commonly it is mainly from the inferior vena caval blood. The explanation that we offer is the anatomic relationship of the opening of the foramen ovale, pointing, as it does, toward the inferior vena cava.

These phenomena are illustrated (Fig. 4) by dye curves obtained after injections into the superior and inferior vena cava in two patients; the one on the left shows no shunt from the superior vena cava and a minimal shunt from the inferior vena cava; the curves on the right show a minimal shunt from the superior vena cava, and a larger, moderate-sized shunt from the inferior vena cava.

The distortion of the *disappearance slope of the concentration* of dye in the presence of *left-to-right shunts* has already been mentioned. When dye is injected directly into the pulmonary artery, the lengthening of the disappearance slope may be more nearly accurately shown as being composed of an early recircula-

VENTRICULAR SEPTAL DEFECT-LEFT
Left to right shunt

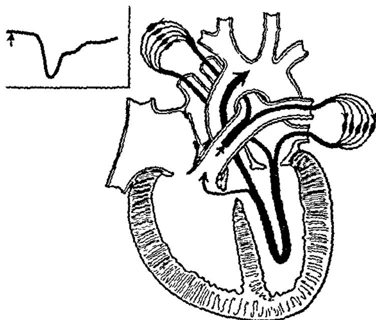


Fig. 5

tion peak, the *secondary hump in this curve* being related to the dyed blood, *shown by the small arrow in the diagram*, circulating through the lung the second time and then entering the aorta (Fig. 5).

The actual records of such a case are shown in Figure 6, wherein the rapid circulation peak, *coming at approximately 10*

to the shunt, into the right ventricle, the dye appears early in the aorta and the characteristic double-hump dilution curve of the right-to-left shunt is obtained. If the shunt had been at the atrial level, the dye curve after injection into the right ventricle would have been normal and that after injection into the atrium, abnormal.

In many patients in whom the classic clinical and laboratory findings of atrial septal defect are made, in addition to the large left-to-right shunt, small transatrial right-to-left shunts have been demonstrated. This shunt may arise from a small portion of

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INFERIOR AS OPPOSED TO SUPERIOR VENA CAVA
IN CASES OF ATRIAL SEPTAL DEFECT

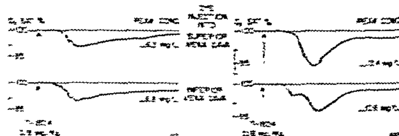


Fig. 4

superior vena caval blood crossing to the left atrium, but commonly it is mainly from the inferior vena caval blood. The explanation that we offer is the anatomic relationship of the opening of the foramen ovale, pointing, as it does, toward the inferior vena cava.

These phenomena are illustrated (Fig. 4) by dye curves obtained after injections into the superior and inferior vena cava in two patients; the one on the left shows no shunt from the superior vena cava and a minimal shunt from the inferior vena cava; the curves on the right show a minimal shunt from the superior vena cava, and a larger, moderate-sized shunt from the inferior vena cava.

seconds after the main peak, is shown when the injection is made into the pulmonary trunk, but the effect is partially smeared out when the injection is made into the superior vena cava.

In the presence of atrial septal defects, the dye curves after injections into the right and left pulmonary arteries are characteristically different (Fig. 7). The amount of recirculation from the right lung is much exaggerated as compared to that from the

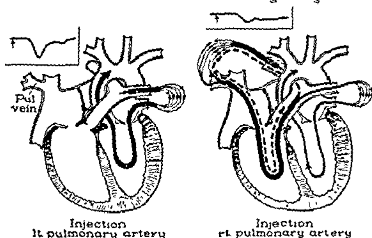


Fig 8

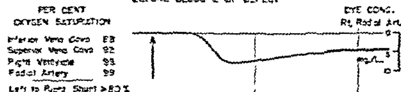
left An injection into the right pulmonary artery labels the blood therein, and the larger amount of blood can be shown to pass through the defect to recirculate, while only a small amount of this blood from the right lung pursues the normal course into the left ventricle. On the other hand, an injection into the left pulmonary artery is followed by a much more nearly normal dye curve, a large amount of blood entering by the normal course into the left ventricle, while only a small amount passes across the defect. The percentage of such preferential shunting varies from case to case.

When the described technics are used, the diagnosis of anom-

alous venous connection of either lung can be made. As an example, let us suppose blood from the right lung is draining into the junction of the superior vena cava and the right atrium, and that the atrial septum is intact (Fig. 8).* If dye is injected into the normally draining lung, a perfectly normal dye curve is obtained. Consider what happens when the injection is made into the right pulmonary artery: the dye has to pass through the lung,

DEMONSTRATION DURING OPERATION OF COMPLETE CLOSURE OF AN ATRIAL SEPTAL DEFECT

(♀–23 YEARS)



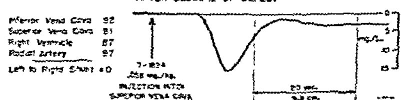


Fig. 9

return to the right side of the heart, and then a portion must pass through the normally drained lung and return to the left ventricle, *then only* to be pumped into the aorta, before the dye dilution record can commence. Thus, in the dye curve, one has a prolonged appearance time, a markedly reduced deflection, and a long, drawn-out disappearance slope, corresponding to the gradual release of the dye from the central pool, as portions

* Reproduced, with permission of the publisher, from: Kirklin, J. W., Swan, H. J. C., Wood, E. H., Burchell, H. B. and Edwards, J. E.: "Anatomic, Physiologic and Surgical Considerations in Repair of Interatrial Communications in Man." *J. Thoracic Surg.* 29:37-49 (Jan.) 1955.

of it are transferred into the left lung with each cycle of recirculation

The tests described usually are carried out in the cardiovascular laboratories, but they may be also performed in the operating room, and a record can be made on portable apparatus. Thus, the results of surgical procedures can be monitored as they are being carried out, for example, the disappearance of a left-to-right shunt can be demonstrated after an atrial septal defect has been repaired successfully (Fig 9)

5

Angiocardiography in Two Planes and with Multiple Frames

JOHN LIND, Stockholm, Sweden

When discussing angiocardiography, one must first state which method has been used. A diagnostic failure with a method taking two pictures per second in one plane does not necessarily mean a diagnostic failure with a method taking a multiple exposure of ten to twelve per second in two planes synchronously.

I am going to present the main characteristics of angiocardiography used in our hospital in Stockholm. Angiocardiography is always combined with catheterization and has not been used alone. Our procedure is: first, a thorough clinical examination, then the catheterization, and thereafter we decide how the angiocardiography is to be carried out—whether by intravenous angiocardiography with visualization of the heart and the large vessels, or by a partial or selective angiocardiography where the contrast medium is injected at a strategic point.

Further, the angiocardiographic method is in a state of rapid development. What is not possible today may well be possible within the year.

The main characteristics of the angiocardiographic method

are a simultaneous recording of the ECG and synchronous radiography in two planes, rapid exposure, and contrast injection depending on the expected diagnosis The synchronous ECG permits each pair of pictures to be accurately timed in the cardiac cycle, permitting a more dynamic interpretation.

With modern equipment, it is possible to change the exposure rate in taking angiocardiograms If one is interested especially in the visualization of the right heart, a rapid rate is used at the beginning, and then, for several seconds, a slower rate, eventually increasing the rate of exposures toward the end

There are some advantages in using synchronous bi-plane angiocardiography—less risk is involved, only one injection is needed, interpretation is aided because one can localize the contrast medium in the heart exactly, and three-dimension evaluation is obtained

By using a rapid exposure rate, one can catch each heart chamber in the extreme phases of its cycle, for example, the left atrium in maximal diastole, and then in systole, and one can judge its size better when one knows the extremes during the cardiac cycle.

Rapid exposure rate at the same time gives more anatomical information, as well as a concept of the dynamic action of the heart and the mechanism of the contraction of different parts of the chamber. Last but not least, an estimate of the mode of passage of the contrast medium—the course, velocity, and distribution—is obtained In a way, it gives a photographic recording of a dye curve. You can see whether there is a normal or an abnormal dilution, and, as I have said, all accumulations of dye are well shown.

More anatomical information may be gained in some cases of Fallot's tetralogy with a pulmonary stenosis, because in ventricular diastole, the pulmonary artery seems to be of normal calibre, but in ventricular systole, there is a significant narrowing.

The same is true in some cases of Fallot's tetralogy. There may be a normal-looking pulmonary artery in ventricular dia-

stole but during ventricular systole one sees the post-stenotic dilatation.

In patent ductus arteriosus, one can diagnose the left-to-right shunt practically always, in children at least, by rapid angiocardiography During ventricular diastole, no contrast medium reaches the pulmonary artery from the right ventricle, but the shunt from the aorta, in which no dye remains, causes a dilution of the contrast medium during ventricular diastole only in the pulmonary artery and not the right ventricle. This is significant for aortic septal defect or patent ductus.

At the end, in a ductus case, the concentration of contrast medium is higher in the aorta than in the pulmonary artery, and one gets a re-opacification or a prolonged visualization of the pulmonary artery In the left anterior oblique, one can see the left atrium, left ventricle, and aorta, but not the pulmonary artery. But in the following ventricular systole, contrast medium is coming from the aorta, visualizing the pulmonary artery, only to disappear again in the following ventricular diastole etc. When one sees this phenomenon repeating itself, over a number of cycles the diagnosis is more reliable

These are the indications for different methods of instillation of the contrast medium Intravenous injection is used when the whole circulation is to be studied. For partial or selective angiocardiography, depending on the expected diagnosis, one can put the tip of the catheter in different places. In anomalous venous return, it is put in the pulmonary artery and injected through it or, perhaps, if one knows from which side the venous return comes, in the right or the left main branch of the pulmonary artery In atrial septal defect, the idea is to put the catheter in the left atrium, but if one does not succeed, then in the pulmonary artery. We have used injection in the right ventricle, especially in the diagnosis of the position and function of the pulmonary conus and pulmonary valves in tetralogy of Fallot or isolated pulmonary valvular stenosis. Injection through the right ventricle gives more satisfactory results in such cases.

The image amplifiers of Phillips take 32 to 48 pictures per

second. By using this image amplifier, you can take 1500 pictures with an x-ray dose to the skin of only 5 R. It has the very great advantage that you can view the screen at the same time as you take your angiocardiograms. You can correct the position and see what has happened in the meantime

PART II

Memorials

Chairman:
HOWARD B. SPRAGUE, Boston, Massachusetts

6

Frank Norman Wilson: Physician, Scholar, Friend

LOUIS N. KATZ, Chicago, Illinois

It is only fitting that this formal memorial to Frank Norman Wilson should be part of a World Congress of Cardiology, the first held in the United States of America It is also appropriate that it should be shared with one to Willem Einthoven, since they, more than anyone else, helped reduce electrocardiography to a mathematical science

Dr Wilson believed in international meetings for the exchange of scientific knowledge He attended the First and Second Inter-American Cardiological Congresses in Mexico City and the Third in Chicago Unfortunately, he was too ill in 1950 to go to Paris to the First World Congress, but was vitally interested in it. Death came suddenly to Dr Wilson on September 11, 1952, shortly after the Fourth Inter-American Congress in Buenos Aires. He died of an acute coronary thrombosis, after almost four years of illness I remember the sadness with which his Latin-American colleagues received the news I recall the respect and affection expressed at the memorial meeting held in Lima, Peru, a meeting which must have been many times repeated throughout the world We shall miss Frank Wilson, but his work and his spirit go on

6

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Dr. Wilson's career reminds me of a passage from Herman Melville's book, *Moby Dick*, which I quote in part: "The straight warp of necessity, not to be swerved from its ultimate course...; free will still free to ply her shuttle between given threads; and chance, though restrained . . . by both, . . . by turns rules either, and has the last featuring blow at events."

It was fate that Frank Wilson should appear on the scene after Willem Einthoven, and during the zenith of the careers of Lewis in London and of Rothberger and Wenckenbach in Vienna. It was chance that led him to be assigned to Colchester, England, during World War I, to work with Sir Thomas Lewis and thus establish a lifelong friendship of mutual benefit. It was chance that led Dr. Walter Hewlett, his chief, to ask Wilson to become responsible for assembling and using the electrocardiograph newly acquired by the department. It was destiny that brought Dr. and Mrs. Wilson together, a companionship which gave him support in his dedicated work and in the avocations which were his relaxation.

But through it all, free will was paramount. It was evidenced by his determination, by his concentration, by his probing after fundamentals and his search to understand the tools with which he worked—be they physical, biological, or mathematical. He taught himself mathematics and electrophysics, since he needed the knowledge for his studies. He had a logical mind and a brilliant one, so that his self-determination bore abundant fruit. This brilliancy was apparent early. On graduating from medicine, he was elected to the honorary medical fraternity, Alpha Omega Alpha, for high scholastic standing. His thoroughness is best exemplified by the fact, testified to by his lifelong friend, Dr. Samuel A. Levine, that when Wilson was introduced to chess during World War I, a game he had never played before, he quickly acquired several books on the subject and began studying them diligently. Before long, he became a master of the game and could outplay the other members of his group.

Dr. Wilson was not deterred by the fact that what he did was not popular. He was not impressed by the need to work in a fashionable field. He pursued his chosen way, without deviation,

to the goal to which he had dedicated his life's devotion. His early years were thankless, lonely, and unrewarding, in terms of the interest and awareness of the rest of the medical profession. But he carried on, firmly convinced of the importance of his work. His ultimate triumph is a tribute to his foresight and perseverance.

Dr. Wilson was a great physician, kindly and humane, and his personal experience with his own protracted illness gave him even greater insight into disease, for, as Thomas Carlyle puts it in Characteristics, "The healthy know not of their health, but only the sick . ." Dr Paul S Barker, in a memorandum kindly prepared for me, said, "Dr Wilson was an eminent physician. His work as a consultant was notable for thoroughness, clinical acumen, sound judgment, and the warm, human qualities which endeared him to his patients His retentive memory served him well in collecting clinical observations . . ."

It was Wilson's recognition of the frailties of clinical diagnosis that led him, as a physician, to devote so much time to an evaluation of the factors which contribute to electrocardiographic contour It was the fact that he was a physician that led him to be, in the words of Dr Franklin D Johnston, "an exponent of sane and conservative interpretation of electrocardiograms . . [that] made him acutely aware of the many things apart from heart disease that may alter the records, and he (Dr Wilson) often commented that the more a physician knows about electrocardiography, the more conservative his interpretation of the records will be" This lesson from Dr Wilson could be emulated by all of us It is, as much as anything, the motivation for the symposium which is to follow.

Dr Wilson was more than a physician He was a scholar In his chosen field, electrocardiography, he was a "selfmade man" He was a product of the Midwest. Here he was born, near Detroit; here he died, 62 years later, on his farm near Stockbridge, Michigan. Here he was educated in grade and high school, in college and medical school Here he joined the medical faculty of his alma mater, the University of Michigan at Ann Arbor, to become one of its most distinguished members. Here in the

old Heart Station, under a wooden stairway which led to the main medical amphitheatre, he did his best work The absence of windows, and the noise and dust raised by the tramping feet of medical students pasing overhead, did not disturb Wilson's power of concentration.

Eventually, a new university hospital was built and Wilson obtained a laboratory commensurate with his worth. To this shrine came many distinguished scientists and physicians, and a multitude of disciples and students to sit at the feet of this master in electrocardiography Among his distinguished disciples are Drs. George Herrmann, Shelby Wishart, Paul Barker, Robert Bayley, Nelson Cotrim, Hans Hecht, Roberto Menezes de Oliveira, Roberto Scarsi, Franklin Johnston, Herman Erlanger, Victor Alzamora-Castro, Fernando Valencia, Charles Kossmann, A. Garrard MacLeod, Demetrio Sodi-Pallares, William Sodeman, Francis Rosenbaum, Ian G W. Hill, and J Marion Bryant. This reads like a list from a *Who's Who in Electrocardiography* His influence was spread even further because he gave an annual postgraduate course in electrocardiography in which he stressed the fundamental basic concepts so helpful to an aspiring student. This was a popular course, noted for its clarity of presentation and illustration.

Dr. Wilson lectured far and wide and received many honors. He was invited to lecture not only in this country but also in Argentina, Brazil, Chile, Great Britain, Mexico, Peru, and Uruguay He derived much pleasure from these visits and from the renewal of old friendships and the formation of new ones. He was deeply appreciative of the hospitality and courtesies shown him and the many honors accorded him

He gave the annual Henry Russell Lecture at the University of Michigan in 1940, one of the highest honors bestowed by the faculty upon one of its members He was the recipient, in 1951, of the Gold Heart Award of the American Heart Association, its highest honor In 1950, he was elected an honorary member, together with Sir John Parkinson, of the International Cardiological Society at the inaugural meeting in Paris In one of the two murals painted by the famous Mexican artist, Diego

Rivera, in the entrance hall of the Instituto Nacional de Cardiología de Mexico, Frank Norman Wilson's portrait is included among the illustrious of all time.

The University of Michigan can indeed be proud of him, because, as Sir William Osler states in *Aequanimitas*, "The great possession of any university is its great names. It is not the 'pride, pomp, and circumstance' of an institution which brings honour, nor its wealth, nor the number of its schools, nor the students who throng its halls, but the men who have trodden in its service the thorny road through toil . . . to the serene abode of Fame . . ."

Dr Wilson, in the words of Dr George R. Herrmann, was a "typical absent-minded professor, totally absorbed in his thoughts and often losing his way in walking or driving to his home . . ." He never practiced showmanship. He was not interested in medical politics, wide acclaim, publicity, or personal aggrandizement He was a quiet, deliberate, and modest worker. He took great pains to present his views clearly and precisely Besides, he had a wide range of interests which included photography, history, political philosophy, and ornithology.

But, above all, Frank Norman Wilson was a friend. He included people from all walks of life among his friends—colleagues, students, patients, and neighbors He was loyal and generous, although intolerant of mediocrity and carelessness His friendship was not syrupy, saccharine or satiating, but, like a rare dry vintage French wine, it was sharp, bracing, and satisfying.

There was a lighter aspect of his character that only his intimates had the privilege to know. In Dr Levine's words, "Many friends have shared the rustic warmth of Wilson's farm Here, one could see beyond the academic and erudite exterior a lively interest in wholesome pleasures Worn clothes, new ideas, simple living, searching exchange of thoughts were part of the fare . . . Nothing would delight him more than to have the group join in song around the piano and try out their lusty voices in singing favorite tunes."

This is the man who contributed so much to scientific knowl-

edge, whose exposition provides a firm and fundamental foundation in the field of electrocardiography, whose work has given us insight into the contour of the ventricular complex and its changes with conduction disturbances, with infarction of the myocardium and with hypertrophy of the ventricles. His concept of electrical position and of the ventricular gradient are fundamental to our understanding of electrocardiography. It is no more than proper, then, that the central terminal which he arranged should be called the Wilson terminal.

What more need be said about Frank Norman Wilson—Physician, Scholar, Friend? What more than to quote the famous German writer, Wolfgang Goethe, who says in *Das Gottliche*·

Edel sei der Mensch,
Hilfreich und gut!
Denn das Allein
Unterscheidet ihn
Von allen Wesen
Die wir Kennen.

7

The Fiftieth Anniversary of the String Galvanometer: Willem Einthoven

H. A. SNELLEN, Leiden, Netherlands

It is now a little over fifty years ago that Einthoven published his string galvanometer in an article entitled, "Ein Neues Galvanometer"

The construction of this new galvanometer may truly be called the initiating event of modern electrocardiography which has grown into a vast clinical science Einthoven himself contributed largely to this development even before the construction of his galvanometer

His interest in electrocardiography started very soon after his appointment as professor of physiology in the University of Leiden in 1885 at the age of 25, a very unusually young age even in those days It appears that the great physiologist and ophthalmologist, Donders, Einthoven's teacher, was mainly responsible for this Einthoven had not yet obtained his medical license on receiving the appointment, although he had written a thesis on stereoscopy by means of difference of color. In his *inauguration speech, Einthoven recalled, while addressing the* students, that he was one of them only a very short while ago, and he proposed to be their fellow student again, to learn from nature whenever his experience and knowledge would fall

short. He added he expected to continue to do so during his whole life. This statement seems very closely related to his answer many years later to interviewers, who asked him how he managed to get the results which brought to him the Nobel Prize. He then replied, "I just wanted to know."

Einthoven must indeed have had a real thirst for knowledge combined with a clear and methodical mind and a meticulous care for all details. At the same time, he showed a great feeling for separating essentials from nonessentials, as is apparent both from his many publications and from his personal annotations now preserved in the museum for the history of science and medicine at Leiden.

This feeling was largely based on a sound knowledge of physics and mathematics which he had acquired by his own efforts without special education. On one occasion, he even felt it necessary to free himself from all other work in order to study mathematics. He used to ask the experts in the university, who included such famous men as Lorentz, only after he had made his own calculations first

It seems to me that Einthoven showed the best qualities of a specialist, in the modern sense, by concentrating on a limited subject, mainly, though not exclusively, electrocardiography, and then studying this from all possible angles, thereby encountering many different problems of a predominantly physical nature, which he studied methodically and fully. These problems include the visibility of very thin threads, the means of damping galvanometer oscillations, the protection of an oscillatory system against extraneous mechanical disturbances, the electrical impedance of the human body, and many others.

Einthoven was vividly interested in all technical refinements which could improve his results, but had very little technical skill himself. If, however, his highly skilled technicians were at a loss while looking for the cause of instrumental disturbances, he was often able to direct them to the source of the trouble, because he knew and understood all technical details.

During the first ten years or so, Einthoven used the Lippman capillary electrometer for electrocardiographic studies and, as

early as 1893, also for phonocardiographic researches. This instrument, however, which had also been used in the earlier investigations of Waller, proved too slowly reacting although fairly sensitive Einthoven set out both to improve the instrument and to study its physical characteristics, thereby devising a way to correct mathematically all the curves which he obtained.

At this point, he introduced the designation of the waves of the electrocardiogram by the letters, P, Q, R, S, T, proposed a uniform technique of registration speed and of calibration, and began to use the first of the standard leads applying electrodes to both hands. The necessary mathematical correction of every curve, however, was a laborious method, and Einthoven felt the necessity to devise an instrument which would combine the greatest sensitivity with high speed of indication.

To this end, he studied the coil galvanometer of Desprez d'Arsonval, and calculated that the smallest possible number of windings on the coil, that is, one half of a winding, would be best for his purpose. His next conclusion was that a very light straight string should be used, and so he constructed his metal-coated thin thread of quartz, fabricated at first by shooting a melted piece of glass with a bow and arrow

Now, in later years, the string galvanometer was improved in various ways, and after Einthoven was able to employ very thin threads, less than one-tenth of a micron, he used the string as a mechanical resonance system for sounds, and also, by placing a very short string in a high vacuum, as an electrical resonance system for radiotelegraphy, even receiving signals from the then Dutch East Indies in collaboration with his son, an electrical engineer. This is the vacuum galvanometer.

By freeing the string container from air, the sensitivity of the galvanometer was, of course, greatly increased, but here Einthoven encountered the ultimate limit of sensitivity in the shape of oscillations of the string due to Brownian molecular movements.

Of Einthoven's many contributions to clinical electrocardiography, I wish to mention only his investigation on the

changes in the electrocardiogram due to respiration and to changes in the position of the body. These observations could be explained by introducing the conception of the electrical axis, the position and length of which could be computed by using the scheme of the equilateral triangle.

This conception, cautiously applied to a particular and rather simple problem, was later used by Einthoven for calculating the changes of the heart vector in size and direction during the heart cycle, thus constituting a precursor of vectorcardiography. It also proved a source of inspiration to many subsequent workers, among whom Frank Wilson should be mentioned first of all But, on the other hand, it caused much confusion and misunderstanding among those who did not fully grasp the limitations of the extremely simplified character of the triangle scheme, the limitations of which Einthoven himself was very well aware.

Many other investigators have contributed to the development of electrocardiography after the construction of the string galvanometer, and Einthoven was as ready to admit this as he expected others to give him full credit for his own work. In particular, he realized the importance of Sir Thomas Lewis's studies and, in accepting the Nobel Prize in 1925, he mentioned the very great share which Lewis took in the development of electrocardiography, and added, "I doubt if I would have had the honor of standing here before you without his valuable contributions."

PART III

Electrocardiography and Ballistocardiography

Chairmen:
LOUIS N. KATZ, *Chicago, Illinois*
ISAAC STARR, Philadelphia, Pennsylvania

8

Vectorcardiography and Electrocardiography

A Discussion of Two Cases

CHAIRMAN LOUIS N KATZ We had selected four unknown cases, but time will permit us to present only two. Each of the panelists, has received either one unknown vectorcardiogram, or one unknown electrocardiogram, upon which, as Dr. Puddu put it, "he will be examined." The panelists do not know anything about the clinical story and they know nothing about the necropsy findings. They are reading these recordings objectively In order that the audience may participate, slides will be projected so you can see how you compare with our panelists.

In selecting these cases, I have had the able assistance of Dr. Charles Kossmann and Dr. George Burch, who will act as Co-Chairmen, each taking responsibility for one of these cases. Dr. Burch supplied one of the cases to be presented, and Dr. Arthur Grishman, the other. Dr Grishman will be examined on Dr. Burch's case. This session is to test the practicality of the two types of records rather than the theory behind their use.

Dr. Jouve of France, who was to have been one of our stereovector experts, unfortunately could not come. His colleagues, the stereovector experts, will be handicapped by their smaller number.

The panelists are Dr. Pierre Duchosal of Switzerland, an

outstanding exponent of stereovectorcardiography, Dr. J. B. Milovanovich, originally of Yugoslavia but now a thoroughgoing Parisian, Dr. W. Den Boer of Holland; Dr. Louis Wolff of Boston, Dr Arthur Grishman of New York; Dr. George Burch of New Orleans, Dr. Charles Kossmann of New York; Dr. Gordon B. Myers of Detroit; Dr. Conger Williams of Boston, Dr. Vittorio Puddu of Rome, Italy; Dr. Marcel Segers of Brussels, Belgium, Dr. Max Holzmann of Zurich, Switzerland, whose book on electrocardiography in German is one of the classics; and Dr. Demetrio Sodi-Pallares of Mexico City, also author of a standard textbook on this subject in Spanish.

We will start with Case P. N. (Fig. 10A), and we will first call on Dr Wolff, who will speak for two minutes on the diagnosis of the case.

DR. LOUIS WOLFF: I believe this vectorcardiogram is abnormal, because the spatial position of the QRS and T loops is outside normal, and because there is a marked divergence of the QRS and T loops. Furthermore, it appears, though one cannot be certain from the record, that the initial forces are oriented to the left, posteriorly and superiorly, and the S-T junction is displaced to the right, anteriorly and down.

Having said that the vectorcardiogram is abnormal, I have said all that I am sure about in this record. There are at least two reasons for this One, I have no personal experience whatsoever with the reference system used in obtaining the vectorcardiogram and, in reproducing the vectorcardiogram, the initial forces are not clearly delineated. Nevertheless, I will attempt to make a more definitive diagnosis than that it is just an abnormal tracing

The three conditions which I think should be considered are left ventricular hypertrophy, left bundle branch block, and myocardial infarction. In left ventricular hypertrophy, the spatial loop is considerably wider than it appears to be here and, in many cases, the terminal or late forces are markedly displaced to the right, posteriorly and superiorly. The S-T segment junction in isolated left ventricular hypertrophy, at least with the reference system which we employ in patients

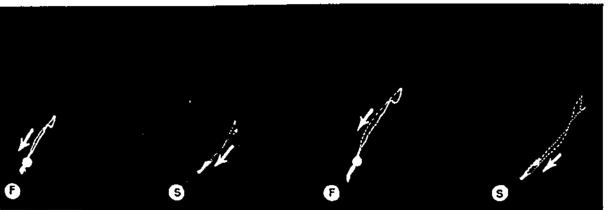


Fig. 10 A. Vectorcardiogram for Case P N

who are not receiving digitalis, is displaced to the right, posteriorly and superiorly. For these reasons, I will exclude left ventricular hypertrophy as an isolated condition.

Now, one of the things that strikes one in looking at this curve is its very close resemblance to the curves published by Burch and which are designated as left bundle branch block, left ventricular hypertrophy, prolonged QRS interval, and diffuse myocardial disease. It is even tempting to make this diagnosis, the resemblance is so close.

However, we must be true to ourselves, so to speak, and what we have learned with an entirely different reference system leads us in another direction. In the first place, if we make a diagnosis of left bundle branch block, we do not feel that we can recognize myocardial disease or myocardial infarction, except in rare cases in which there is, perhaps, massive infarction Furthermore, the most important diagnostic feature, the one that we depend upon greatly, is the orientation of the initial forces, which, in left bundle branch block, is to the left, anteriorly and down. Here, you remember, the initial forces are directed to the left, posteriorly and superiorly. Therefore, I have to exclude a left bundle branch block, as attractive as this diagnosis seems.

That leaves us, then, with myocardial infarction. I may briefly say that the initial forces are directed posteriorly and to the left. That is abnormal, when there is no left bundle branch block, and that we have seen in proved cases of anteroseptal myocardial infarction. The continuation in an exaggerated way of the early forces, posteriorly, we have seen in proved cases of anterior myocardial infarction. And, finally, the initial forces point superiorly, which we have observed repeatedly in proved cases of inferior wall myocardial infarction. The S-T segment junction located to the right, anteriorly and down, is consistent with the combination of anterior and posterior wall infarction.

My diagnosis, then, is myocardial infarction of the anterior wall and posterior wall of the left ventricle, and of the interventricular septum.

CHAIRMAN KATZ: For those of you who are not familiar with the modality that Dr. Burch uses, he wrote me that F represents the frontal plane and S the sagittal plane. The transverse axis connections of the frontal plane are made so that when the right arm is negative and the left arm is positive, the deflection goes to the viewer's right or the patient's left, that is, towards the left-arm electrode. In the vertical direction, when the central terminal is relatively negative, the deflection goes downward In the sagittal plane, when the back electrode is relatively positive with respect to the central terminal, the deflection is toward the back. The polarity is the same for the central terminal and foot as it was in the frontal plane

The two sets of curves are different amplifications, that of the pair on the left being 1 millivolt = 19 mm, and that of the pair on the right being 1 millivolt = 29 mm The interrupted line is arranged so that the blunt end indicates the direction of the movement of the loop The time interval is such that each interruption is 1.06 second. The reference frame employed is the equilateral tetrahedron as used by the Burch school

Dr. Kossmann and I thought it would be much simpler not to use all the vectors that Dr. Burch sent us, and we assume responsibility for this limited exhibition of material Dr Grishman, can you agree or disagree with the diagnosis of Dr. Wolff?

DR ARTHUR GRISHMAN: I agree with the basic diagnosis of Dr. Wolff As we see this vectorcardiogram, it has the following characteristics to me: It is superiorly oriented It is strongly posteriorly oriented. Furthermore, it has no anterior deflection. It has an interruption and some peculiarity at its tip, and it is a periphery of the QS loop. Furthermore, the QRS loop does not close There is an S–T vector present, which is directed predominantly, I assume, anteriorly and inferiorly.

I do not believe this is a left bundle branch block because there is some irregularity and some slowing, but this happens to be only in the initial phase of the QS vector and not in the terminal phase I believe we have an infarction of the an-

teroseptal portion, because there is no anterior deflection present.

I furthermore believe that we have a *diaphragmatic infarction* present at the same time, because the superior orientation is more than what we usually expect, even with the tetrahedral system, where the orientation is more often superiorly oriented in left ventricular hypertrophy, it is more here than we should suspect in left ventricular hypertrophy alone.

It is hard to tell from the left projection alone if the S-T vector is one mainly associated with left ventricular hypertrophy or if there is an acute injury vector, in addition. The T loop does not give the impression that there is an acute myocardial infarction present; at least one cannot interpret it from the record, because the T loop is just spindle-shaped and long.

I would therefore assume that this patient has left ventricular preponderance, and I do not like the term, "strain" or "hypertrophy"; anteroseptal infarction, and some additional myocardial injury which is probably located inferiorly. That is all.

CHAIRMAN KATZ: We will now ask the electrocardiograph team to accept the challange. Dr. Sodi-Pallares will speak first on what the electrocardiogram shows. This will now be projected (Fig. 10*B*).

DR. DEMETRIO SODI-PALLARES. I believe this tracing shows first, auricular fibrillation, second, probably left ventricular hypertrophy, since the lowest end is plus 21 mm., the QRS is located at $-60°$, and there is a delay of the "intrinsic" deflection in leads V_5 and V_6; third, there is decrease and altered morphology of QRS in leads V_5 and V_6, plus the *duration* of QRS which, in some leads, is 0.11 second, and this suggests some degree of left bundle branch block (we call this second degree of left bundle branch block). Fourth, there is probably *myocardial infarction* of the lower third of the intraventricular septum. There is an R wave in V_1 which disappears in leads V_2, V_3, and V_4, and also there is a Q wave in leads V_5 and V_6, in the presence of incomplete left bundle branch block. If *infarction* exists, it must reach the anteroseptal region of the heart, and may reach the posterior-inferior aspect of the heart,

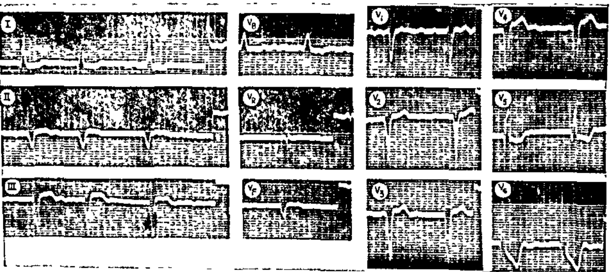


FIG 10 *B* Electrocardiogram for Case P N.

since there are QS complexes in leads 2 and 3. I believe also that infarction extends to the free left ventricular wall of the apex of the heart, but does not reach the lateral portions of the same wall. We have seen that some of these infarctions are complicated with ventricular aneurysms.

Fifth, there is a sort of myocardial injury in the higher lateral portion of the free left ventricular wall. This injury could be related, for example, to digitalis action. That is one possibility. It could also be related to another possibility, that is, chronic coronary insufficiency. I do not believe that infarction is the cause of this

CHAIRMAN KATZ: Dr. Gordon Myers, do you agree or disagree?

DR. GORDON B MYERS. I agree essentially with the diagnosis that has already been made. The slow ventricular rate in this auricular fibrillation suggests a superimposed digitalis effect, which is also supported by the S–T segment configuration, particularly in lead V_5. The presence of a definite R wave in lead V_3, the QS complexes in leads V_2, V_3 and V_4 point very strongly towards anterior infarction—as Dr. Sodi-Pallares said, an infarct in the anterior apical third of the left ventricle, going into the septum The classical confirmatory sign is to obtain an abnormal QR complex in an adjacent lead, indicating subendocardial extension of such infarct.

Leads V_5 and V_6 show a definite Q wave, but that Q wave is normal in duration from onset to nadir, and also in its relative amplitude in comparison to the R wave. The late R wave and the late "intrinsic" downward stroke, as Dr. Sodi-Pallares just mentioned, point towards left ventricular hypertrophy, and, thus, my feeling of the findings in leads V_5 and V_6 is left ventricular hypertrophy, which is usually associated with an infarct.

We would have taken lead V_4 in the fourth interspace in order to demonstrate an abnormal QR complex, by which I mean a Q wave which measured 0 03 second or more from onset to nadir, and is more than one-quarter of the amplitude of the succeeding R. That would have helped confirm our

diagnosis of an anterior septal infarction in this person with left ventricular hypertrophy.

CHAIRMAN KATZ: Thank you very much, Dr. Myers, for putting it so well and so briefly. Now, Dr. Vittorio Puddu, do you agree?

DR VITTORIO PUDDU. Yes, I agree generally This is a tracing with fibrillation and a median ventricular rate of 60 There is a very strong left shift of the electrical axis, which speaks for preponderance. I am not sure if we have the right to speak of bundle branch block or only of some intraventricular block, connected with the hypertrophy.

I agree about the infarction. I do not know if the infarction has extended to the lateral portion of the apex or, perhaps, more probably superiorly because I see a big R wave in leads V_5 and V_6. I think that the infarction is in the interventricular septum, partly in the posterior aspect of the heart, because of the QS in leads 2, 3, and V_6. It is also in the anterior septum and upper part of the lateral left ventricle because of the very deep T wave, digitalis-like, in lead V_6, and the Q wave in leads V_5 and V_6 The elevation of the S–T interval in leads 2 and 3, and the depression of the junction in lead V_1 and some lateral precordial leads point to digitalis or relatively recent subendocardial lesions still showing injury effects.

To summarize, therefore, there is present auricular fibrillation, left ventricular hypertrophy, multiple or diffuse infarction, recent injury, and subendocardial or digitalis action.

CHAIRMAN KATZ Dr. Burch will now read the clinical history. He and I believe the panel has done very well.

DR. GEORGE BURCH: Well, briefly, the clinical data are as follows. This was a 65-year-old male, who came into the Charity Hospital in New Orleans on January 14, 1951, and died two days later. Apparently, he came in with severe chest pain of 12 hours' duration.

This man had been in congestive heart failure with dyspnea as his main complaint at the Charity Hospital clinics since 1949. He received the usual therapy for congestive heart failure. He had a rumbling diastolic mitral murmur and a to-

and-fro aortic murmur. His serology was weakly positive on one occasion and, I think, strongly positive once, and then negative afterwards.

He was found to have massive cardiac enlargement, and his blood pressure at that time was 110/58 mm. Hg. He had a prostatectomy done in 1950. About a year later, and the day before admission, he was suddenly seized with dyspnea, generalized weakness, severe ache in the base of his chest which rapidly changed and was referred down to the substernal region of the chest, associated with profuse perspiration.

On examination, he was found to be seriously ill, particularly confronted with respiratory distress. His blood pressure was 100/70 mm. Hg, his temperature was 99°F., his heart rate was 100, and he was reported to have irregular rhythm by one observer. No murmurs were heard at that time. Signs of congestive heart failure were present. He had a slight leukocytosis with a 76 per cent polymorphonuclear leukocyte count and a sedimentation rate of 51. His past history was essentially irrelevant, except for the fact that he had attacks of rheumatism forty years before. He died suddenly while on the wards of the hospital.

CHAIRMAN KATZ: Do Dr. Wolff or Dr. Grishman wish to change their minds before they get a copy of the necropsy? They say, "No." Do the electrocardiographers wish to change their minds? Dr Sodi-Pallares says "No," Dr. Puddu says "No," and Dr Myers says "No." Dr. Burch will now let you *in on* the secret of what was found at necropsy.

DR BURCH. The autopsy findings are very briefly summarized here. This man had a very large heart, weighing about 800 gm There was an aortic and mitral valve lesion, associated primarily with fibrous thickening, without calcification. He had two infarcts, he had an anteroseptal infarct involving the intraventricular region and penetrating into the septum. That was an old infarct. He also had an old infarction which was posterior in location and seemed to go around the apex. In addition to that, the circumflex coronary artery was filled with a fairly long, rather recently developed thrombus. There was

softening and yellow discoloration at the center with some red discoloration at its periphery, in one region of the heart, located primarily in the free wall of the left ventricle in the region of that circumflex vessel

The pathologist told me that he felt that this patient had old infarction, with a recent occlusion of the circumflex vessel. The infarct was extensive, and it involved the subendocardial and subepicardial surfaces It was a transmural type of infarct, with a lot of fibrous scarring, as well as the recent lesion

CHAIRMAN KATZ: Now, will you discuss the lessons to be learned from this exercise so far?

DR. BURCH: I think, as I listened to the interpretations of Doctors Wolff and Grishman, that they did extremely well. There may be some debate, I guess, in the mind of Dr. Wolff—and he can correct that—as to whether there was left ventricular hypertrophy in association with these infarcts Both of them localized the infarction, I think, very well

The electrocardiography group, I would say, hit the nail squarely on the head. They were able to identify the auricular fibrillation, which the vectorcardiogram, obviously, did not present They recognized the digitalis effects in the electrocardiogram. Whether there was subendocardial disturbance present, or whether these changes were due to digitalis effects superimposed on the infarct, and ischemic effects, I will leave up to the panel to decide. I do not know whether I can decide the question, but it should certainly have been brought up, as the people in the electrocardiography group did

The problem of left ventricular hypertrophy versus some block, so-called second-degree left bundle branch block which Dr Sodi-Pallares mentioned, is something that we debated this morning when we presented some records of left ventricular hypertrophy. In the presence of an infarct involving the septum, Q waves in lead V_1 and lead V_5 and V_6 may be present, with a rather high degree of left bundle branch block. Again, these Q waves may be due to early involvement of the septum.

I do not believe that we ourselves can be sure as to the

nature of the defective conduction in the ventricle, but we do feel that the electrocardiogram did show that, and the vectorcardiogram, we felt, likewise showed some conduction defects. Whether one wishes to call that defective intraventricular conduction (an old term) or whether one wishes to call it second-degree bundle branch block, incomplete, or something else, I do not know. We discussed that this morning, and it is a problem to bring up.

I would say, in conclusion that I presented these tracings to members of our laboratory, independent of the vectorcardiogram, and I have the interpretations of Dr. Ashman, who read the tracings during the routine interpretations on the ward to us, the electrocardiographic interpretations were more extensive and more definite than the vectorcardiographic ones. But, as you saw, the vectorcardiography group did identify the lesions fairly well. I think they ought to comment further as to the relative contributions of these two types of tracings.

CHAIRMAN KATZ: I should like to add my own admiration of the skill with which the stereovector people handled this case. Do you want to add anything, Dr. Grishman?

DR. GRISHMAN: Yes, I would like to add something The S–T vector in this case was definitely located anteriorly. The S–T vector of subendocardial ischemia, although it gives similar deflection for lead I and leads V_5 and V_6, namely, S–T segment depressions, is usually located posteriorly, and posteriorly and superiorly. This vector was directed anteriorly and inferiorly.

I assume that subendocardial ischemia played no role in the causation of the S–T vector. I believe that most of the S–T vector was probably caused by left ventricular preponderance. If digitalis had an effect, or if there was an exaggeration of the S–T vector by an acute coronary injury, I have difficulty in determining it. Of course, not having known that the patient was fibrillating—which, from this type of tracing, I cannot tell—I cannot quickly jump to the conclusion that this patient was digitalized.

I think Dr. Sodi-Pallares, if I may say so, said this patient is fibrillating and is digitalized, and I think that sequence in

thought is quite proper, and we usually do it when we see auricular fibrillation. We usually suspect that the patient, if he is treated at all, is somehow controlled with digitalis or other medication.

CHAIRMAN KATZ: Dr. Wolff, would you like to make a statement?

DR WOLFF. According to our observations, it seems to me that the S–T vector is characteristic only when one is dealing with single conditions, let us say, either an anterior myocardial infarct or a posterior infarct, or left ventricular hypertrophy or right ventricular hypertrophy. When multiple infarcts exist and when both left and right ventricular hypertrophy are present and, in addition, if the patient is on digitalis, then, of course, what one observes in the vectorcardiogram is the resultant forces as they are influenced by the various factors. In this case, then, I think all we can say is that the S–T vector is consistent with all the lesions that were found in this case

CHAIRMAN KATZ· Dr. Myers, do you wish to comment?

DR MYERS: This case merely illustrates a point that we made many times in the past, that most of our errors are errors of omission. You will note that from the electrocardiogram it is quite possible to make the diagnosis of auricular fibrillation, digitalis effect, and anterior septal myocardial infarct in left ventricular hypertrophy. We all missed the recent terminal posterolateral infarct, and the reason we missed it was because we did not have leads in the proper place. We should have had leads high in position V_6, V_7 and V_8 to pick up the high posterolateral infarction. I think we could have made the diagnosis if we had those leads.

The QS in lead V_6 and in leads 2 and 3 is probably merely a manifestation of the septal infarct in a heart which lies horizontally, and therefore potential variations are transmitted to the lead.

CHAIRMAN KATZ: Dr. Puddu, do you wish to make any comment?

DR. PUDDU: No.

CHAIRMAN KATZ: Dr. Sodi-Pallares?

DR. SODI-PALLARES: Just to say that I agree with Dr. Grishman, because the vector of S–T is located forward and inferiorly, it was located on the frontal plane, close to 120°, and a very clear forward location. That means that the injury, at least from the electrocardiographic point, is located high in the lateral portion of the ventricular wall, and I do not see how that can be correlated with the first infarction, with the infarction in the lower portion of the ventricular septum

CHAIRMAN KATZ I think this closes the first part of the panel. I think you will agree with me that the performers were magnificent and deserve a hand.

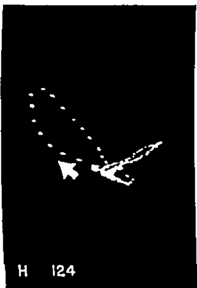
The second case, Case M. D (Fig 11A), will now be shown: we will have a new team to recite. Since this is Dr. Grisham's case, he will not recite. Dr. Duchosal, will you summarize the vectorcardiogram?

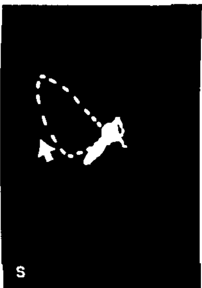
DR PIERRE DUCHOSAL· I think I can epitomize what it is This is taken with the technique of Grishman, a modification of Duchosal.

Description of the case· QRS loop duration is about 0 08 second The main portion of the loop is located in the right superior posterior quartile, or towards the right shoulder. The small initial portion of the QRS loop is directly opposed to the former The T loop is directed downward, posteriorly and to the left It is a narrow, elongated loop of rather great amplitude The P loop is doubtful, the S–T segment, not visible.

Interpretation Since the initial portion of the QRS loop is directed to the left and anteriorly, while the major portion is backward, to the right shoulder, this is strongly in favor of a massive right ventricular hypertrophy. The position of the T loop in relation to the QRS loop is similar to that commonly seen with right ventricular hypertrophy. Right ventricular hypertrophy of this extreme degree is most likely of congenital origin.

It is regrettable that the submitted document contains an obvious error in the indication of the RST loop location. The rotation of the R plane is incompatible with the reverse rotation of the S–T plane. In addition, the arrows definitely in-





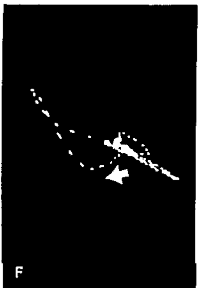


Fig 11 A. Vectorcardiogram for Case M D

dicate that the large portion of the loop occurs first, which is a misleading indication. This is not a likely possibility since this would signify a large anterior or lateral infarction, and the T loop is incompatible with this situation.

All this has been confirmed and clarified by consulting two previous original publications by Grishman and his associates, where the entire case is reported and published, so that the unknown, as ingeniously announced by our Chairman, is in reality well known.

CHAIRMAN KATZ: Will Dr Den Boer, without reference to the fact that this case was presumably once published, read the stereovectorgram and arrive at a diagnosis?

DR. W. DEN BOER. I might, first, point out that I am not entirely satisfied with the theoretical approach underlying this system as a reference frame. Still, I believe that this is a case of very strong preponderance of the right ventricle, although one cannot be absolutely sure how the QRS loop is inscribed. It might be first directed to the right posteriorly and inferiorly; it might be directed superiorly and nearly vertically, or to the left anteriorly and slightly inferiorly. The last probably applies.

In any case, the main direction of the QRS vector is oriented very strongly to the right, whereas the T loop is oriented essentially opposite the QRS loop. In the horizontal projection, the QRS loop is inscribed clockwise. There is a smooth curve. There is no evidence of conduction delay.

I do not believe this is right bundle branch block. As I had to make my diagnosis, I thought this was probably a case of congenital heart disease, and I have only seen it in interatrial defect, and then I looked in the book by Dr. Grishman and I saw that this case was published. That was somewhat of a disappointment.

CHAIRMAN KATZ. Well, sometimes we pull tricks, and this case may not be the case that you and Dr. Duchosal think it is.

Dr. Milovanovich, you wrote me that you did not like: (1) the orientation, and (2) the lead connections. Furthermore, you wrote that you would have preferred to have the device

that you have described, whereby you can get the heart rate with the VCG. With these omissions, will you please tell us what you think this VCG shows?

DR. J. B. MILOVANOVICH: Well, when I got this material, about a month or two ago, I was just leaving my flat, and I had a look at these nice records. I put them in my car and went away. I must say I was rather annoyed. Usually, we are not supposed to say, by glancing at a vectorcardiogram or electrocardiogram, how old a patient is purported to be. Here, there was no mention of the age.

I was still driving and thinking of this picture as it was, a picture of unusual shape. We never see such a picture in normal or even pathological cases in adults, so I supposed that this would definitely be a child and probably under 10 years old.

As there is no sign of what we have described in 1948, I do not think that it would be a large communication between the right and the left ventricle in this case, so I do not think it is the tetralogy of Fallot, but, rather, a trilogy of Fallot, that is, a high degree of right ventricular hypertrophy, stenosis of the pulmonary artery, and most probably communication between the two ventricles. It happens sometimes, however, that in old people, we may have such a picture in a person who has pulmonary emphysema, but in such cases, the T loop is not so highly developed, so I still believe that it is the diagnosis I just gave you.

CHAIRMAN KATZ Dr. Max Holzmann will lead off with the diagnosis from the electrocardiogram (Fig 11*B*).

DR. MAX HOLZMANN: The ECG represents a P wave of normal duration; the P is rather high and broad in lead I, without an inverted segment in leads V_1 to V_2. The P–Q interval is normal. The ventricular complex depicts a normal QRS duration of 0 08 seconds. There are very small Q waves in leads 2 and 3. The S wave prevails in leads 1, 2, V_6 and in the standard chest leads, with the R wave prevailing in leads V_{R6} to V_{R3}. The mean QRS axis in the frontal plane falls in a sector of −150 to −180°, and is rotated backward.

The S–T segment is hardly elevated in leads 1 and 2. The

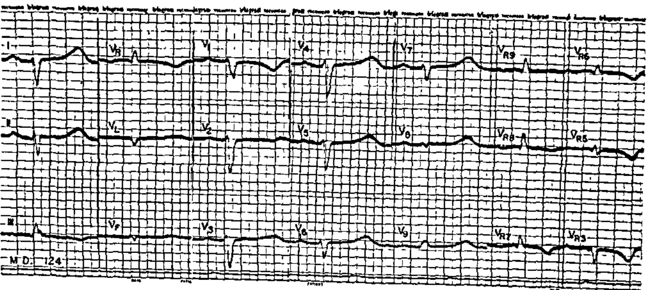


Fig. 11 B Electrocardiogram for Case M. D.

T wave is generaly opposite to the main QRS vector The biggest T axis looks to the left, down and back, because in the frontal plane sector, it is between 0 and 30°.

Conclusions. There must be a severe hypertrophy of the right ventricle, with damage of the right ventricular myocardium. The pattern is similar to that found in rare cases of severe mitral stenosis or in cases of congenital heart disease, with right ventricular strain, as in Fallot's disease or pure pulmonic stenosis. The P wave configuration in the limb leads might be considered to be in favor of the severe mitral lesion, but the chest leads do not support this view Therefore, I feel that this is rather in favor of a congenital origin

I should like to join in the opinion expressed by Dr. Milovanovich that this group of tracings is not completely satisfactory because the strips are cut too short Therefore, it is not possible to relate the Q–T interval to the heart rate, and we cannot make up our minds about the Q waves.

CHAIRMAN KATZ Dr Segers, do you agree with Dr. Holzmann?

DR. MARCEL SEGERS Yes, the major interpretations correspond here to a marked deviation of QRS in the limb leads, with large T_1 and T_2 with the axis deviated toward the left. In the left precordial leads, the S wave remains very deep until in lead V_7, on the opposite side There are R waves in the precordial leads, lead V_{R6} to V_{R9} This pattern indicates marked ventricular hypertrophy, with an abnormal position of the heart, following focal right ventricular block of minor degree, in the posterior part of the ventricle. But there are no evident signs of cor pulmonale or of mitral stenosis, so my conclusion is congenital heart disease with right ventricular hypertrophy.

CHAIRMAN KATZ And now, Dr. Williams, do you agree with your colleagues?

DR. CONGER WILLIAMS I do. I think that the diagnosis of right ventricular hypertrophy is quite obvious from a glance at the tracing I agree also that there are conduction defects, both intraventricular and probably auriculoventricular as well, since

the P–R interval, in some leads at least, appears to be as long as 0.30 second. For this, too, I think, I would be in favor of a congenital lesion; that is, I am speaking of the auriculoventricular conduction defect in association with right ventricular hypertrophy and an intraventricular conduction defect.

Perhaps we could be more certain if this were complete heart block, because we do frequently see complete heart block in association with congenital defects such as ventricular or low auricular septal defect.

For these reasons, I think it very likely that congenital heart disease is present. Perhaps another less likely possibility is that *of a generally destructive infiltrative lesion of some* unknown source in a patient who already has right ventricular hypertrophy.

CHAIRMAN KATZ. I inadvertently sent this series of records to other members of the ECG panel, and Dr. Sodi-Pallares tells me that he and his group spent hours unraveling it, and I think it only fair to ask him if he agrees with his colleagues on the panel

DR SODI-PALLARES: We also believe, from this tracing, that it is, first, right ventricular hypertrophy; in fact, the QRS is located near plus or minus 180°, and pointed slightly backward, and the T is at most 10°, almost diametrically opposed.

Second, we believe there is important right auricular dilatation. As you can see, there is a small Q wave in the entire right hemithorax. We have described that kind of pattern mainly in right auricular dilatation.

We thought of three main diagnostic possibilities. We thought, certainly, of congenital heart disease, but we also thought of chronic cor pulmonale, and we thought of the possibility of displacement of the heart.

Now, the elliptical axis of P is around plus 30°. That has been described also in our department as a common finding, and we have called that the "congenital" P wave. Because of that, we were inclined to the possibility of congenital heart disease. We thought mainly not of pulmonary stenosis *although*

we did believe it could be pulmonary stenosis, but of interauricular communication.

Also, we must rule out the single ventricle because in the single ventricle, as it has been described, there is a smaller heart and waves in the precordial leads than is shown in this tracing

CHAIRMAN KATZ· Dr. Myers, do you want to say something at this point?

DR MYERS We made no extended study of this case Our big problem was to identify the ventricle responsible for leads V_{R6}, V_{R7}, V_{R8}, and V_{R9}, all of which showed a late "intrinsic" deflection. We had to consider a right ventricular hypertrophy, on the one hand. We also considered the possibility that the heart was in an abnormal position, and these were coming from the left ventricle, due to backward rotation on the transverse axis, along with right rotation, as described by Dr. Kossmann, and we concluded, as have the others, that this was the result of right ventricular hypertrophy, for a little different reason; namely, that these leads that I have already given, that is, V_{R6} to V_{R9}, inclusive, have, as an initial deflection, a Q wave. The same type of initial deflection is also seen in V_{R5}, V_{R3}, and V_{R1}, all of which show a Q wave. We classed these as leads reflecting predominantly the potential variations of the right ventricle.

In contrast, we felt that leads V_2 to V_7, inclusive, were left ventricular leads predominantly

Having diagnosed right ventricular hypertrophy, we considered causes, and, of course, agree that congenital heart disease has to be thought of in anybody with severe right ventricular hypertrophy. We felt, however, in this case, that the ventricular variation of the left ventricle referred to a relatively wide area, and therefore favored some acquired heart disease, either cor pulmonale or the fish-mouth type of mitral stenosis, which will give a wider dissemination of the left ventricular potentials.

CHAIRMAN KATZ. Dr. Kossmann wants to ask a question.

DR. CHARLES KOSSMANN: I would like to ask a question of

Dr. Sodi-Pallares. Would you please clarify, Dr. Sodi-Pallares, your statement about the appearance of a Q wave in leads from the right side of the chest, when there is right atrial *dilatation, and, second, would you please elaborate on the* occurrence in this patient of late intrinsic or RS deflection, if you like, on the right side of the chest posteriorly, as opposed to the absence of such a deflection on the right side.

DR. SODI-PALLARES: When you work directly on the heart—and I am making reference not only to the dog's heart but also to the human heart—in cases of right ventricular hypertrophy, it has been found that in all the epicardial surface of the right ventricle, there never is a Q wave. You get the Q wave only when you go directly to the epicardial surface of the right ventricle very close to the right auricle.

I do not know if Dr. Kossmann has read our papers about the meaning of the Q wave in leads V_1 and V_2, and also in the right precordial leads. We have also found right auricular dilatation, certainly; but almost always there is, at the same *time, right ventricle dilatation. I remember a case with tricuspid* stenosis, which showed a QR complex in leads V_1 and V_2, and there was a very big right auricle and a very small right ventricle.

We must not forget that we are speaking of potential variations, not of potential action. I know, certainly, that Dr Grishman explained the meaning of that tiny Q wave in right ventricular hypertrophy as a diminution of the conductivity of the Purkinje fibers, but we do not believe it is so We believe, in these cases, there is a big right auricle, and that transmits the potential variations that exist in the upper part of the intraventricular septum. In the upper part of the intraventricular septum, you get Q, more Q, and bigger waves, mainly in some degrees of complete or incomplete right bundle branch block.

CHAIRMAN KATZ. Dr Duchosal, do you want to make any comment?

DR. DUCHOSAL· The only comment that I should like to make is concerning the right preponderance, which has been con-

sidered to be possibly either due to congenital heart disease or rheumatic heart disease· mitral stenosis for instance. Indeed, it is very rare to have such a great right shift of the QRS loop in cases of mitral stenosis, even in very stenotic cases, and this appearance of the vectorcardiogram is very much in favor of the very big enlargements which can be found only in congenital heart disease.

CHAIRMAN KATZ. Dr. Kossmann wants to ask you a question, Dr. Duchosal.

DR. KOSSMANN: Dr Duchosal, I think you said that the duration of the QRS was 0 08 second. Dr. Williams pointed out, on the conventional leads, that it was in excess of 0.1 second Since you, presumably, have had the advantage of seeing this case in advance—you said it was published—would you like to review the duration of the QRS in the vectorcardiogram? There does seem to be a discrepancy here

DR. DUCHOSAL· You know, this is a nice question, because the figure is very poor, and I did my best to measure the timing only in the vectorcardiogram, indeed, and I was very much embarrassed because the two mistakes I told you about are very gross mistakes indeed

The direction of the arrows is wrong On the H plane, they are in the reverse direction to the S and F, and if you have good eyes, you will immediately see that it is wrong This is a very misleading indication.

The second very misleading indication is the start of the loop, which is wrongly indicated in the frontal plane It starts from zero, going directly to the right, which is wrong It goes from zero to the left, and it is misleading because these arrows are badly placed

In the second publication of the case, in the *American Heart Journal,* there was a little arrow pointing first to the left, but that was taken away in this presentation.

CHAIRMAN KATZ· Now, having had all the dilemmas, we will have the clinical story and the necropsy findings, which Dr. Kossmann will present.

DR. KOSSMANN This patient was 32 years old, female. She

was complaining of fatigue and cyanosis and dyspnea. She was known to be a blue baby since birth, but she had no symptoms until she was 8 years old, when she began to complain of fatigue. These became intensified so that at the age of 29, her first admission to the hospital was made.

At that time, in 1948, the patient was cyanotic; she had clubbing of the fingers and toes. The heart was not enlarged on examination and no thrills were felt. Rhythm was regular. A–2 was flat, louder than P–2. A harsh systolic murmur was heard best in the left sternal border, in the third left intercostal space.

Important laboratory data. hemoglobin, 13 gms.; red blood cells, 7.7 million, venous pressure, 10 cm. of water, ether circulation time, 5 seconds.

There was also a catheterization study at this time, not listed on the slide, and it was noted that the oxygen content of the blood taken from the right atrium varied from time to time. The color could even be seen to be different with different specimens. X-ray of the chest showed globular configuration of the heart, with slight enlargement to the right, and diminished pulmonary vascular markings Arterial oxygen saturation, 64 per cent. Angiocardiography was done.

Course: A Blalock-Taussig procedure was performed. Postoperatively, a continuous murmur appeared in the left interclavicular region, and the pulse pressure increased. Cyanosis decreased somewhat. The patient's exercise tolerance improved, allowing her to return to work.

There was a second admission, three years after the operation, for recurrence of the former complaint and, in addition, she had attacks of dizziness. A physical examination was similar to the previous admission, except that a continuous murmur, with a faint diastolic component, was heard in the left interclavicular region.

Laboratory tests: hemoglobin, 16 *gm.; red blood cells,* 7.8 million; hematocrit, 63 per cent. The chest x-ray was similar to the previous one. The arterial oxygen saturation was 60 per cent. Incidentally, a catheterization done at this time did

not show very much difference from the first one, except that *this* time the catheter was placed in the right ventricle where it had not been placed before, and the pressure in that chamber was 200 mm. Hg systolic and 10 to 20 mm. Hg diastolic.

The patient had several episodes of numbness and twitching of the extremities. She died suddenly, following a convulsion, after mild activity, and before cardiac surgery which had been contemplated could be performed.

CHAIRMAN KATZ: The panelists did so well, and because time is passing so quickly, we will go at once to the necropsy findings, which will be presented by Dr. Kossmann

DR. KOSSMANN: The right atrium was markedly dilated and hypertrophied. The foramen ovale was widely patent and, in effect, was a real atrial septal defect, 2 cm. in diameter. The right atrial endocardium was thickened, smooth and free of thrombus; the tricuspid valve had a low-lying ring and a rudimentary posterior leaflet; the right ventricle was hypertrophied, its thickness being up to 0 8 cm; pulmonary valve ring, 1.3 cm. in diameter; marked pulmonary valvular stenosis due to fusion of the cusps; poststenotic dilatation of the pulmonary artery.

There was no patent ductus. The left heart was normal. There was an old anastomosis between the left subclavian and left pulmonary artery, but it was not functioning. It was represented by a fibrous band between the thrombosed subclavian and pulmonary artery. There was dilatation of one bronchial artery. There was an old infarct of the spleen and kidneys and chronic thyroiditis.

CHAIRMAN KATZ: Will you summarize the lesson to be learned from this case, Dr. Kossmann?

DR. KOSSMANN. I think both groups of the panel—the vectorcardiograph members and the electrocardiographic members—did very well. Dr. Duchosal, of course, knew what the case was We did not try to trick you. The case was published before. However, we did give the panel some data that were not published.

I think that in this instance, we have an electrocardiogram

and a vectorcardiogram which could go with several anatomical situations, giving considerable enlargement or hypertrophy of the right side of the heart, but, as you heard from the panelists on both sides, there were little hints to indicate that the most likely thing was congenital heart disease, simply because the electrocardiographic findings suggested rather extensive right ventricular hypertrophy, and in acquired heart disease one does get hypertrophy, but it is almost never massive.

I do not know which of the two sides of the panel did the better. I think they both did very well, but, again, Dr. Duchosal pointed out that there were technical difficulties. These are very real and must be contended with if one is going to do vectorcardiography on a clinical basis with the equipment now available.

On the other hand, with the conventional records, these intervals were easy to pick out. As Dr. Holzmann pointed out, we did not have enough records on the slide to make it possible for him to decide the importance of the duration of the Q–T interval. That, of course, is a small technical difficulty due to mounting. It was done for convenience. We could have given him the heart rate if he wanted it

I think, in conclusion, that there is no great difference in this particular case between the two sides of the panel, although we do have a few advantages in favor of the conventional method, because of the technique and because of the timing that it is possible to get from it, which is more accurately than from the vectorcardiogram.

CHAIRMAN KATZ: Dr Den Boer, do you have any comments to make?

DR. DEN BOER· No, thank you.

CHAIRMAN KATZ. Dr. Milovanovich, do you have any comments to make?

DR. MILOVANOVICH: I find, as you realize, that these pictures were not taken very well, technically. It would be rather difficult to say where the P loop is, how large, how long is its duration, what about the QRS duration, and so on. I think that

the vectorcardiogram, when it is performed, ought to be completed by recording a vector on a moving paper.

CHAIRMAN KATZ: Dr. Duchosal?

DR. DUCHOSAL: I have nothing to add.

CHAIRMAN KATZ Dr Williams, have you any comments to make? Dr. Segers? Dr. Holzmann? Dr. Burch wishes to make a comment.

DR. BURCH. I should like, before we adjourn, to comment about Dr. Sodi-Pallares's recognition of enlarged right atrium His associates have been pointing out that electrocardiograph sign, and, as you saw, he recognized from the electrocardiogram the large right atrium I think we should not pass that by without indicating our appreciation of that accomplishment

CHAIRMAN KATZ. I should like to have Dr. Kossmann make one or two remarks before I close this session.

DR KOSSMANN: I should like at this time to ask a question of all the members of the panel I think one may get the impression from a session of this type that one can make precise anatomical diagnoses from the electrocardiogram That is true in some instances, and these were two extreme instances where the correlation between the electrophysiological and the anatomical findings was very good But I think that it should be pointed out that the vectorcardiographer and the electrocardiographer who are doing routine work will more often not be able to say with any degree of precision what the anatomical diagnosis is

With those introductory remarks, I should like to ask the panel to answer by a show of hands, if they wish, the following question, and, at the end of the question, I will give the alternatives. What is the entire panel's opinion concerning the common practice of making anatomical diagnoses from an electrophysiological method? The three possibilities are there is no correlation between the two, there is a very high correlation between the two, or, there is a moderate correlation [The show of hands was inconclusive.] Well, at least, we are able to demonstrate that on this point there is a difference of opinion among the panel members.

CHAIRMAN KATZ: I think we should show our appreciation to the second half of the panel for their splendid performance as we did for the first.

I am sure that all of you have shared with me the pleasure of an exercise which is not dependent upon a man simply getting up and making a formal presentation, where nobody can disagree with him. In this session, we have demonstrated that perceptive physicians can deduce what the electrocardiogram and vectorcardiogram will show, before the clinical history and physical findings are given. I think we should continue this kind of practice at future congresses. If you agree, will you so indicate by letter to our Secretary-General, Dr. Pierre Duchosal.

9

Ballistocardiography

A Panel Discussion

CHAIRMAN ISAAC STARR During my medical lifetime, a healthy change in attitude has come over medical faculties in the United States. When I was a medical student, about thirty-five years ago, many of my professors, with conspicuous exceptions, talked from the standpoint of authority. They regarded themselves as having the last word in knowledge of their subject, and we poor students could do no better than to sit at their feet and memorize what they had to say. But, unhappily, a portion of what they had to say has proved, by the passage of time, to be erroneous, and I now look back on some of my teachers of that era as conceited, opinionated, and overconfident.

I mention this past era to call your attention to the fact that a change in atmosphere has taken place in most North American medical schools, and that the typical American professor is far more humble than his predecessors I am sure that this change is in the right direction

This change in attitude has led to the search for new and better methods of presentation of medical subjects, not only to students but also to audiences such as this one, who have attained competence and distinction in this field. One of the new forms of presentation now being widely used, the form we pro-

pose to employ here, is called the panel discussion. The officers in charge of this Congress have not asked me to give you a lecture on the ballistocardiogram, but to gather together persons with experience in the field, to present their views on various aspects of it and then to be prepared to answer whatever questions may arise either from other members of the panel or from the audience. Therefore I am hopeful that this session will submit to this distinguished audience not what purports to be an authoritative presentation of the subject but rather a discussion of the ballistocardiogram in which various points of view are presented. For I do not expect the group that I have gathered to agree fully either with each other or with myself on many topics. I expect that many differences of opinion will be brought out, and I shall regard such differences of opinion not with concern but as evidence that the field is in a truly healthy state and ready for further advance I believe that promising fields such as this one will advance, and not by pronouncements from self-constituted authorities but by hard work to bring out facts so obvious that all must agree, and by hard thinking about what such facts mean, and by a proper respect for the opinion of other workers in the field

Before starting, I must thank the members of the panel for consenting to take part, and I must express regret that I was unable to secure the services of Dr. William Dock of New York, who has done so much to make these records more available generally to physicians. He was invited among the first, but other plans prevented his taking part with us today.

I must also apologize to Dr. von Wittern that his name has somehow escaped printing in the program. He was one of the first I asked to take part in the panel

Something inherent in the panel technique is the danger that discussion and questions may become so unrelated to each other that no real view of the field as a whole is secured by the audience. My opinion was that the audience would probably need some extended view of the field, and, to guard against this danger, I have asked some of the members of my panel to give short presentations on aspects of the subject that have particu-

larly interested them. These presentations will take up much less than the time allotted to us, leaving the remainder for questions and answers. I have invited other persons besides these speakers to ask and answer such questions and to take part in the discussion. Of these, Dr. John L Nickerson, a physiologist of what might be called the physical school, is the author of a ballistocardiogram of the nonresistant table type, Dr Talbot is a physicist who has interested himself in the perfection of ballistocardiographic instrumentation, and Dr. Brown is a clinician who wrote the first monograph published in this field.

As the more formal part of the program is given, I hope that you will write down any questions you would like to ask any of the speakers and hand your questions to me as the session goes on We have made arrangements for having them translated should you care to give them in your native language These questions may be addressed to a particular speaker or to the panel as a whole, in the latter case, I shall call upon a particular member of the panel whom I judge to be best equipped to answer.

Now, having sat through all the previous presentations today in this room, I should like to point out some similarities and some differences to what has gone on before. I was most impressed, as I am sure many of you must be, with the surgeons, and the thing that particularly interested me in that meeting was the question they were asking themselves, "What lesions that the patient suffers from are anatomical and therefore corrected by surgery, and what are not anatomical?"

It is the nonanatomical, or shall we call them the functional, or, perhaps better, physiological lesions of the cardiac contraction that the ballistocardiogram is aimed to detect.

Second, with regard to the electrocardiographers who have preceded us in such great numbers, I cannot help calling a little attention to the great influence that they have put on making anatomical diagnoses. I say that to call attention to the difference between the electrocardiogram and the ballistocardiogram. I doubt very much if the ballistocardiogram is going to be of great help in making diagnoses of the anatomical type. It

may be of help in some instances, I agree, but the main interest in the field is something totally different. It is helpful in determining aspects of physiological cardiac function about which the other methods simply do not supply us with any information.

When my students ask me the difference between the electrocardiogram and the ballistocardiogram, I always point out one simple thing. If you take an electrocardiogram, then exercise, and then take another one, the electrocardiogram is affected little, if at all But if you take ballistocardiograms, the ballistocardiogram after exercise is hugely increased in amplitude in a striking fashion. In other words, when the heart beats more strongly, the ballistocardiogram detects the difference. The electrocardiogram does not do so. It is that kind of physiological difference, differences in the strength of the heart's beating, that we are aiming to make available to clinicians. Needless to say, there are many difficulties and I hope these will be plainly brought out.

I am first going to call on Dr von Wittern, a physicist by training, who has interested himself in the physical aspects of the problem and whose ideas and technical skill may well lead to improvement in our instruments.

DR. WOLF-WITO VON WITTERN: The ballistocardiogram is a record of the oscillatory motion of the body or of a platform on which the body lies. The forces which produce this motion are reaction forces generated by the acceleration or deceleration of masses within the body, that is, the masses of the blood and muscle tissues.

The motions of the body and platform depend not only on the force but, at the same time, on the mechanical system of body and platform In such a system, the pattern of motion, the ballistocardiogram, will in general be different from the patterns of force, and only under certain conditions will the patterns of force and motion be equal. A certain force pattern can produce almost any pattern of motion depending on the structure of the mechanical system

To be able to draw conclusions from the ballistocardiogram on circulatory events, the system must be so designed that its

motion is at any time proportional to the force. Periodic patterns of force or of motion can be presented as sums of sinusoidal force or motion patterns of various frequencies—in our case, the frequency of the heart rate and its integer multiples. Force and motion pattern will be equal when equal sinusoidal forces of all the frequencies contained in the pattern produce equal sinusoidal motions synchronously. To determine the behavior of the body-platform system at various frequencies, it is necessary to know the mechanical properties of body and platform

It was possible to show by measurements that the body, lying on a rigid surface, can be represented, at least for frequencies up to 15 cycles per second, by a mass connected with its support by the elasticity and the internal frictional resistance of the external tissues

At one particular frequency, the so-called natural frequency of the system, a given force acting upon this mass—we are talking only about the body system now—will produce excessively large amplitudes of motion, depending on the frictional resistance. This effect is called "response." The natural frequency of the body system was found to be 3 to 4 cycles per second

The platform system represents a similar system, at least for low frequencies, made up of the platform mass and elasticity and frictional resistance of its connection with the ground.

With the combined body-platform system are shown displacement, velocity, and acceleration of the platform caused by forces of various frequencies acting upon the body mass

We find that these functions have extreme values at f_1, the lower system frequency, and at f_2, the upper system frequency Except when these frequencies are close to f_B, the natural frequency of the body, f_1 is determined by the combined mass of body and platform and the elasticity of the platform suspension, while f_2 is determined by the platform mass and the elasticity of the external body tissues.

Let us now consider the response curves which we obtain when we change the mechanical constants of the platform If we increase the stiffness of the platform suspension, we obtain

the case of the "high frequency platform," and f_1 increases more and more. With infinitely large stiffness, it finally reaches f_B, 4 cycles per second, about the natural frequency of the body. This at the same time is the case of the body lying on a rigid surface or the case of the Dock method.

In case of a very stiff platform, the damping of the platform is ineffective and the resonance at f_1 is damped insufficiently only by the body tissue

With the high frequency platform and with the body on a rigid surface, we can expect a displacement record proportional to the force, up to about 5 cycles per second, but this record will be distorted by after-vibrations because of the insufficient damping We work, in this case, in this range here. In the case of a very stiff platform, it goes up to body frequency.

If the stiffness of the platform is decreased, we obtain the case of the "low frequency platform" and f_1 decreases. The resonance at f_1 can be damped by a damping of the platform. There is practically no limit for lowering f_1 and in Talbot's "mercury bed," where the body floats on mercury, f_1 is practically zero

Now, let us consider changes of the platform mass. If we diminish the mass of the platform, f_2 increases and the damping of the resonance at f_2 caused by the body tissues becomes more and more effective When the platform mass is equal to or smaller than about one-tenth of the body mass, this damping is sufficient and, with a platform mass of about one-thirtieth of the body mass, f_2' is raised to about 8 times f_B or 35 cycles per second

By combination of very small platform mass and a very small platform stiffness, it seems possible to obtain a flat response of acceleration. We work, then, in this range. This range is then extended from zero up to infinity, possibly, and this means we have a flat response of acceleration. The acceleration record of the low frequency bed and the displacement record of the high frequency bed should actually render the same results, if the response curves are flat in the same frequency, so it would be possible to obtain a record, maybe, up to 35 cycles per second.

It must be stated, however, that the representation of the body by a one-mass system is justified by measurements only up to 15 cycles per second

The elastic coupling between vessels and body mass has also not been considered, and its influence certainly increases with higher frequencies. We therefore can expect a closer connection between the blood motion and the ballistocardiogram only if we cut off, by means of a filter in the recording system, frequencies larger than 15 to 20 cycles per second.

If we assume that the moved blood mass is constant during one heart cycle, then the acceleration record of the low frequency platform can be interpreted to represent the acceleration of the blood, the velocity record, the velocity of the blood, and the displacement record, the displacement of the blood

There will probably be useful information in the frequency range above 20 cycles per second Unless, however, a flat response in this range is really secured, such information can be used only on a statistical basis

CHAIRMAN STARR Dr von Wittern has, I think, ably given you what might be called the theoretical approach to the subject It is no criticism of Dr von Wittern and other physicists to point out that their expectations concerning the vibration properties of the body have been derived chiefly from experiments performed on inanimate objects, such as steel springs and oil damping, and that predictions and theories based on such results might need considerable modification when applied to body tissues This thought, plus the very obvious but unfortunate fact that my own training in physics stopped at a very elementary level, so that I lack the background to do a first-class job of theoretical physics, has prompted me to seek information about the fundamentals of the ballistocardiogram by another type of attack—the direct experiment.

In these experiments, a great many others took part The fact that I shall not mention their names does not detract from any indebtedness to them.

The type of experiment we finally adopted was determined by the belief that since the amplitude of the ballistocardiogram

was probably related to force, force being, by definition, mass × acceleration, we must devise an experiment in which the acceleration of blood issuing from the heart could be estimated. The experiment we finally adopted was as follows:

A cadaver is placed on a ballistocardiograph. A large glass cannula is inserted through the heart into the aorta and tied in, and another similar cannula is tied into the pulmonary artery. Both cannulae are connected by glass tubing to large syringes whose constricted ends have been removed so that blood flows frequently into the tubing. In contact with the plungers of the syringes is a screen with a slit, through which a light beam records the position of the pistons at each instant and so the cardiac output, let us say, at each instant of systole is recorded. We also record blood pressure in both the aortic arch and femoral artery, as well as the ballistocardiogram, on the table; the resistive table on which the subject lies.

Inflow is through a tube from a bottle, and can be stopped by clamping the tube. As the intra-arterial pressure falls away, as it does in diastole during life, a heavily padded mallet is released and, falling of its own weight, strikes the pistons and simulates systole. In the early days of this work, we used water or saline as perfusing fluid but later we learned how to use blood, securing the blood by saving that about to be discarded from the hospital blood bank because it had been drawn a month previously. The results I mention here will be those secured with blood as perfusing fluid.

In these experiments, we aim to get great variability of conditions. Thus, in the cadavers with perfusion of blood, the stroke volume ranged very widely, over three-fold. The blood pressure varied from hypertensive to shock levels in the different experiments, the duration of ejection corresponded to pulse rates varying from marked tachycardia to marked bradycardia, and records show a great variety in the contour of the ejection curves of the group of experiments in which blood was used. Now, all this variety was obtained purposely, because, certainly, any uniformities which may occur in data as varied as these ought to be thought of as important.

With these data before us, we are in a position to ask ourselves the question, what aspect of cardiac function determined the amplitude of the ballistocardiogram? Expressed in the simplest terms, the results of two important types of experiments come out like this:

When the blow was a large one, a heavy one, the mallet pulled through a large arc, so the ballistocardiogram was large. It represents perfectly the ballistocardiogram secured in healthy people, except for the fact that there is no H wave There is no H wave in any of these experimental ballistocardiograms In contrast, when the blow was a smaller one, the ballistocardiogram is much smaller, the pressures rise less. Also, the ballistocardiogram lacks its I wave

Now, I can summarize some of the main results in simple form. First, when the mallet is dropped through a larger and larger arc, the harder the blow that simulates systole, the larger the ballistocardiogram. We have done this in every subject, with both water and blood experiments, and we simply have never seen it miss, provided conditions are kept constant Everybody, I think, will understand how this was, but not so obvious are the results of an experiment in which we strike the same blow by dropping the mallet through the same arc each time but increase the rubber padding on the mallet, so that the blow struck is softer. The amplitude of the ballistic record diminishes as the blow is softened, even though the mallet drops through the same arc.

In brief, the ballistic amplitude is governed, not by the strength of the blow alone, but also by the way in which that strength is applied When there is little padding, the falling mallet is stopped almost instantaneously, so it delivers its energy in a very short period of time, and the impact is large. When heavily padded, the mallet is stopped gradually, and so it delivers its energy over a much longer period of time, and the resulting ballistic amplitude is much smaller. We have an obvious analogy to driving a nail with a hammer. If steel strikes steel, the nail is driven in, but if you place a pad on the hammer and strike the same blow, the nail is driven in little, if at all.

A more exact method of attack on the genesis of ballistic amplitude is to look for correlations in the data.

The best correlation we have found is between the maximum velocity of the injected blood and the square root of the ballistic I and J wave amplitude The maximum velocity being the result of the accelerations which preceded it, this means to me that the amplitude of the record measures the force of the injection, and therefore, I have come round by an experimental method to the same conclusion that Dr. von Wittern derived in theory. Thus, my results support the view of the previous speaker that the ballistocardiogram taken by recording displacement of a resisted table measures cardiac force primarily.

We can also look for things that improve the correlation, and one of the things is that if you take a factor for body size, the correlation is significantly improved, so that it becomes very high indeed.

Now, the heart being a physiological unit, there are many good correlations between ballistic amplitude and other aspects of cardiac performance. When the heart beats forcefully, the stroke volume is likely to be large, so there is a correlation between ballistic amplitude and stroke volume, a correlation much improved by the addition of a factor for the duration of ejection, as I have had in my earlier experiments.

Our original cardiac output formulae, always regarded as rough methods by us, do not do so badly when tested in experiments such as these, although Tanner's formula, the one I myself preferred, which was adjusted to give results in normal resting subjects corresponding to Fick estimates, gives results which are clearly too high.

By experiments such as these, we can discover whether changes in body properties or physiological functions interfere with an ability to estimate cardiac force from ballistocardiograms. We find that by considering the size of the body, the estimate of force can be improved. It looks as if the bigger the body, the smaller the fraction of cardiac force which escaped to move it, which is perhaps natural. We have also tested changes in the height of the blood pressure, and they make so

small a difference in the estimate of force that they can be neglected The presence of advanced arteriosclerosis probably makes a trifling difference, though our data are still meager on this point.

All this work indicates that by means of the ballistocardiogram, we are certainly on our way to a rough estimate of the strength of the heart's beating, a subject on which doctors have long speculated but about which they have known very little Let me conclude by stressing some qualitative relations, how the form of the ballistocardiogram depends on the contour of the cardiac injection curve, even though this aspect of my work may be very familiar to some of you.

A normal systolic record can be reproduced in the experiments by injections in which the initial acceleration is large On the other hand, if the initial acceleration is small, the ballistocardiogram changes tremendously in contour, becoming what I have called, rather naively, the late downstroke type, and similar to that of ballistocardiograms seen quite frequently in people who by other criteria are thought to have severe heart disease

Injections that are not smooth break up the ballistocardiogram in a wide variety of ways, as also seen in patients with severe heart disease.

If the two sides of the heart are injected asynchronously, abnormalities such as a split J wave result, and this can often be seen in the clinic. The point is that we now know how to reproduce experimentally a great many of the abnormalities, most seen in systole, in the clinic, so we have the right to think that we have a firm grasp on their genesis. The great exception to this statement is concerned with the interesting abnormalities of the H wave at the very onset of contraction Our experiments do not reproduce the H wave, so that our confidence in the interpretations of this interesting part of the record is less than in the other systolic portions

Now, that is all I am going to say about the experimental attack

I am now going to call on Dr. Baker, who is a clinician pri-

marily and who has had wide experience with ballistocardiograms of the resisted table type.

DR. BENJAMIN BAKER: Dr. Starr, it has now been a decade and a half since you revived a dead technique and wisely christened it "ballistocardiography" Among your early observations, there was one which, I believe, has been more responsible than any other for the great interest which has subsequently been shown in this technique That observation was that the ballistocardiograms of patients with coronary artery disease are frequently abnormal when all other measurements of the circulation are normal.

The clinician of that day was as dissatisfied with his management of the coronary artery disease problem as he is today, and quickly adopted this technique in hope of improving his practice

Very soon, numerous observations confirmed those early ones of Dr. Starr and enlarged them. It was very rapidly apparent that many patients with severe coronary artery disease had abnormal ballistocardiograms but others had normal ones. It was also apparent that many seemingly normal people had abnormal ballistocardiograms and other normal people had normal ones.

We have a record from a 55-year-old man with typical angina pectoris. While this patient was quietly lying on the table, he very obligingly developed a severe attack of precordial pain that had all the characteristic features of angina pectoris. Immediately, the electrocardiogram showed the depression of the S–T segment, and the ballistocardiogram deteriorated significantly. His attack was promptly terminated by the administration of nitroglycerin, and a third record was obtained, which is at least *as good as the control record, if not better.*

We have records from a 59-year-old patient, who, very suddenly, without any apparent precipitating factor, developed severe attacks of precordial pain, radiating to the shoulder and down the arm. These attacks were very numerous, and he would frequently have dozens during the course of the night. They were miraculously relieved by nitroglycerin. They never occurred during effort. Actually, while the patient was being

investigated, he got out of bed and, on his own hook, ran up several flights of stairs, to test it out He said the physicians asked him this question so frequently that he wanted to be sure about it.

A hiatus hernia was demonstrated. The attacks occurred almost invariably at night or after a large meal, and if he assumed the erect posture, they would disappear promptly. Nitroglycerin relieved them like magic.

Finally, the patient was operated upon, the hiatus hernia was repaired, and, only ten days after this extensive surgical operation, a new record was obtained. It was at least as good as the earlier record and probably a little better There seems little question but that the attacks were attacks of myocardial ischemia, for there was marked S–T segment depression in the second electrocardiogram which disappeared subsequently

This stimulated us to find out, if possible, how frequently ballistocardiographic abnormality occurred in large numbers of patients with coronary artery disease and in controls We were thoroughly aware, as Dr. Starr has pointed out, that this technique measures physiological phenomena and not anatomical ones, and yet the studies seemed important

The results, with which many of you are familiar for they have been published, are in, roughly, 200 patients with angina pectoris. The results were compared with findings in over 300 normal controls of similar ages. In the fourth decade of life only 45 per cent of the records of patients with angina pectoris were abnormal and no records of normal controls in that decade were abnormal As age advances in both patients and controls, the incidence of form abnormality increases markedly. These abnormalities are significant for all age groups except the eighth decade.

In spite of the fact that these results were interesting, it was hoped that something might be done to make the technique separate the patients from the controls more dramatically. Various stress tests were employed. Some of these were helpful but only one deserves considerable comment, and that is the stress produced by smoking a cigarette, first suggested by Dock and

subsequently investigated thoroughly by several observers.

In approximately 100 normal controls, very few of the ballistocardiograms deteriorated. On the other hand, a very high percentage of the records of patients with coronary artery disease deteriorated significantly when they smoked a cigarette Actually, only about 18 per cent of the records of patients with coronary artery disease remained normal after smoking a cigarette. This test served to separate the patients from the controls in the proportion of 9 to 1.

One would conclude by saying that there is no question whatever but that the ballistocardiograph measures a circulatory dysfunction which is commonly found in patients with coronary artery disease, but is also commonly found in normal controls as they grow older and presumably develop more and more coronary artery disease. This is, however, quite different from saying that a patient with coronary artery disease, who has an abnormal ballistocardiogram, *is* threatened with immediate deterioration of his condition or death. It is also quite different from saying that a normal control, with a normal ballistocardiogram, is secure from the development of it. The facts argue strongly to the contrary.

Everyone knows that the status of the coronary circulation changes rapidly from moment to moment, and it would be too much to expect that a technique could do more than measure the efficiency of it at a given moment. Until further physiological studies and long-term follow-ups brings us complete understanding of ballistocardiography, we urge a conservatism in its use in the clinic, in order that a technique of undoubted value and great promise not be brought into disrepute by making claims for it that are not yet justified by secure facts.

CHAIRMAN STARR: I can assure you, I echo Dr. Baker's wise words. Now, I am next going to call on the last two speakers, who have been using the ballistocardiographs not of the type I commonly employ but of the type which were originated by Dr Dock Dr. Harrison has been, I gather, not quite satisfied with the original technique and has been doing strange things with his subjects, attempting to improve the method. Since all these

methods can be improved, I am going to call on Dr Harrison to tell us a little bit about it.

DR. TINSLEY R. HARRISON: First of all, I should like to say this. whatever value the ballistocardiogram may eventually have will be attributed to Dr. Isaac Starr It is his baby. The rest of us are neophytes in the field compared to Dr. Starr.

Second, much as I fear that agreement among the panelists may detract from your interest, my own little knowledge of the physics of the ballistocardiogram leads me to the same conclusions that Dr. von Wittern has drawn, and, certainly, I would agree with the clinical statements made by Dr Baker on the basis of an experience very much greater than ours.

We regard this as an instrument which has something to do with telling us about the force of the heart, which almost certainly is going to be of practical clinical value ultimately. We take a dim view at the moment of attempting to draw clinical conclusions from it and, so far as I am concerned, the stethoscope and, more particularly, the pencil are still the most important methods of examining a heart

I do not believe that the ideal ballistocardiograph has yet been devised.

It is quite likely that of the numerous different methods being used at present, some will ultimately prove to be more valuable for one purpose and others may prove to be more valuable for another purpose. For example, the technique which we use and which I shall mention briefly in a moment is not a good technique to study disease, because the method does not allow one to record respiratory variations properly, and respiratory variations appear to be quite important as a clue to abnormality The methods which we have used have been devised primarily to attempt to study the genesis of the ballistocardiographic waves and particularly those waves which cannot be reproduced in the cadaver, and about which we therefore have relatively little knowledge at present.

The techniques do seem to allow one to find a very close correlation with certain physiologic events, as recorded by other methods, such as the carotid artery upstroke, the carotid artery

incisura, the various points of change of direction on the electrokymogram, etc.

One can very easily obtain a record which is full of artefacts. The body can get into a certain vibration frequency, and you can get a record that looks a good deal like a sine wave. When the subject has sandbags put around his shoulders and around his feet, the amplitude is less, which one would expect, and the configuration is quite different The J-K downstroke is much less deep, a new little wiggle follows the K wiggle, a K-L upstroke, we call it, followed by a little downstroke, and that little wiggle is absent in the first situation.

It is difficult for me to believe that you can damp wiggles in. You can certainly damp them out. For that reason, we believe the latter situation comes somewhat closer to giving a true ballistocardiogram. The undamped record, incidentally, is something like Dr. Nickerson's records which he has been obtaining for a good many years.

With the subject lying on a putty surface, without any damping at all other than the damping incident to the putty, the K nadir is deep. If the subject is damped by putting sandbags around him, and the sequence changes, the configuration changes. The K nadir becomes much higher, and the K-L-M sequence become much more clear.

If further sandbags are placed around the subject there is little further change. In other words, if one damps the subject, one at first gets a marked change in configuration and then one gets a situation in which little further change in configuration occurs. That makes us think that we are getting somewhere close to the true body wiggles, and that aftervibrations are being largely eliminated.

The chief points I should like to make are: First, I have great faith in the long-range future value of this method as telling us something about the contractile power of the heart. I doubt that it is ever going to tell us anything about the anatomical lesions in the heart.

Second, I do not believe, on the basis of our own experience at least, that the method is yet ready for general clinical use.

That statement must be qualified by admitting that we ourselves have not paid a great deal of attention to that and have not tried deliberately to use techniques that might give the most information in disease.

Third, if one records from the shins, one has to realize that there are certain artefacts induced which might not be present if one is recording more directly from the foot or from the head

Fourth, a considerable part of the ballistocardiogram, as usually recorded, without dampening of the body, appears to represent sine wave oscillations and does not appear to represent physiologic forces.

Finally, by the use of techniques of progressive damping and techniques which change body frequency, such as the use of sand, putty, and additional sandbags, one can obtain a record which has a close correlation with other physiologic events. Whether that is particularly important, I do not know, but it has seemed so to us.

CHAIRMAN STARR: And now for the last of what you might call "prepared speakers," I am going to call on Dr. Mandelbaum of New York, who, following the original Dock technique closely, has had very extensive clinical experience with records of this type.

DR HARRY MANDELBAUM The classic head-foot ballistocardiogram is useful in distinguishing between normal and abnormal traces and in observing the effects of exercise and smoking Routine lateral plane studies are now done simultaneously, as we find it helpful in interpreting properly our recordings of body vibrations set up by cardiac forces We employ two galvanometers and a pulse or sound channel or both, for timing

We have adopted Dr Brown's classification of standards as practicable. However, a Grade I ballistocardiogram is found so often in emphysema and in apprehensive subjects that we regard it as clinically unimportant Grade II changes, if not proven directional in lateral traces, are significant. Grade III and Grade IV traces are regarded as indicating serious abnormality of cardiac function. The finding of a Grade II or worse ballistocardiogram in subjects with no clinical evidence

of heart disease should be regarded as significant at any age.

The importance of the nicotine sensitivity test is widely accepted as correlating with coronary artery disease. I would like to extend Dr. Baker's scope and state that Dr. Scarborough and *his coworkers* at Johns Hopkins, in their study of a group of subjects with coronary artery disease, found significant ballistocardiographic changes *after smoking*. *This occurred nine times* as often as positive Master's two-step exercise tests, and gave no false positives on normals under the age of 30, while the standard two-step exercise test on the same subjects gave over 20 per cent false positive results.

In the last year, we have been combining lateral traces in the upper channel.

We have noticed that occasionally the relationship of the lateral to the longitudinal is what we usually expect in normal patients But under the influence of smoking, in several instances, we have been able to notice notching of the H wave.

The relationship of smoking to hypertension has been well established by Dr. Grace Roth. We have done nicotine sensitivity tests on all our hypertensive subjects and have found a large percentage of positive results.

In angina pectoris, the light exercise test is as important as the nicotine sensitivity test in eliciting abnormalities in the ballistocardiogram While most clinics report an incidence of 60 to 70 per cent abnormal traces in subjects with angina pectoris, the light exercise test will cause deterioration in the ballistocardiogram in an additional 10 to 15 per cent.

In *hypertension*, the resting ballistocardiogram is not conclusive. The light exercise test is a requisite. Bing has shown that exercise relaxes functional coronary "spasm," but in the presence of advanced coronary artery disease or congestive heart failure, this will not occur. Improvement in definitiveness and amplitude after exercise indicates an adequate coronary flow as well as an adequate myocardial reserve.

Dr. Dock has shown that in an occasional hypertensive subject, a decrease in head-foot systolic waves may be paralleled by an increase in lateral systolic waves, and offers as an explana-

tion that direction, not force of ejection, is changing, probably due to tortuosity of the aorta wave.

Large systolic waves may provide the first indication of high cardiac output This is most important when a right-to-left intracardiac shunt or an anomalous venous return to the right auricle give high right ventricular output with normal pulse pressure. High pulse pressure points to high stroke volume, as in aortic regurgitation. In hyperthyroidism, in beri-beri heart disease, in anemia, in arteriovenous fistula, and in Paget's osteitis, the pulse pressure is not notably large, yet, wide amplitude systolic complexes are often striking, even in the presence of failure. Because high output may be corrected by medical or surgical means, its recognition by the ballistocardiograph is of real importance.

Lateral ballistocardiograms bring to light large diastolic waves, due to gallop phenomena, or auricular systole, which may not be apparent on the longitudinal trace These may be striking in myocardial failure and have been noted in constrictive pericarditis.

The ability of the ballistocardiograph to demonstrate the absence of myocardial embarrassment in subjects with healed infarction, with persistent electrocardiographic abnormalities or with bundle branch block, is perhaps of even greater importance than revealing unsuspected, or confirming clinically recognized, coronary artery disease, or showing poor recovery of the heart after infarction Not infrequently, patients have made a good clinical recovery but abnormal head-foot ballistocardiograms persist It has also been noted that many older subjects, with no clinical evidence of heart disease, have abnormal traces. Use of the lateral instrument demonstrates that in many of the latter, and in many asymptomatic cases of healed infarction, especially in those over the age of 50, the force of systole is excellent, although the vector is far more lateral than in young people Thus controlled, the two-plane ballistocardiographic study of patients should now rarely raise suspicion of heart disease when none exists, and often prove that the myocardium functions well in spite of age and previous infarctions.

CHAIRMAN STARR: Now, gentlemen, we have had our chance at the program, and now it is your turn, if you care to take part in it. The panel is up here, ready and willing to answer questions that any of you send up, and if none appear from the audience, we may indeed ask each other questions. Have any come from the audience as yet? No questions seem to have arrived. I am going to start by asking a question of Dr. von Wittern

I am going to say that it looks to me as if some of Dr. Harrison's sandbag tracings were beginning to look a little like the contour of the kind of tracings Dr von Wittern has published. I should like to ask Dr. von Wittern to comment on that, whether he agrees with me, and, if so, what he thinks the nature of the advantage is

DR. VON WITTERN I agree entirely with you, Dr. Starr, on that. I myself wondered that these records were exactly like the ones I took The reason is that by restraining the body by sandbags, the natural frequency of the body is raised, and then we have a flat frequency response up to higher frequencies. At the same time, the damping of the body tissues becomes more effective at a higher frequency, and so we might indeed have a reasonably flat response curve after much higher frequencies than we are used to having in these cases

CHAIRMAN STARR Have any members of the panel any questions they would like to ask other panel members? That not being the case, I have one or two here.

I am anxious to ask Dr Baker what he thinks is causing the abnormality of form that we all are finding in elderly people who have no evidence of any disease by the ordinary methods of testing for such things, in other words, what, then, causes it, do you think?

DR. BAKER: I think, if I could answer that question to the satisfaction of everyone, I would be thoroughly in command of the basic knowledge of ballistocardiography. Obviously, I cannot answer that question definitively.

I think there are, undoubtedly, a number of factors which enter into the abnormal records in older people. Certainly, they are developing progressive coronary artery disease; certainly,

they have very widespread atherosclerosis, changes in the aorta, and large vessels; certainly, they are developing poor abdominal musculature, and we know that abdominal muscle tone has a good deal to do with the form of the ballistocardiogram, certainly, they are developing pulmonary changes and I think that at present it is perfectly impossible to say which of these factors are the truly significant ones In other words, we cannot separate those factors that are truly intracardiac from those which are extracardiac.

While I am here, I should like Dr. Brown, who, I think, has had as much experience, if not more than anyone in this field, to tell us a little about what he considers distinguishing features between normal and abnormal records As far as I know, this is an individual matter In our laboratory, we have one man read all the records, so at least we have uniformity. I think we have a long way to go before we have absolute assurance that we are reading records correctly

DR HERBERT R. BROWN, JR.: Mr Moderator, there are several things, I think, that should be pointed out at this time First, you can use this instrument in the several ways that you have seen You can use it in the acute experiment, for physiologic purposes, or you can use it clinically.

In the second place, you can use it in various different forms of apparatus—the Starr high-frequency table, the longitudinal table, on which my records have all been obtained up to date That is one way. The various portable types of apparatus are another They are not necessarily identically comparable. On the other hand, the abnormalities that accrue in the various forms of apparatus are rather comparable, although not necessarily in timing

As far as abnormality is concerned, if one looks back to the calculations of cardiac output, probably one can find grave discrepancies. On the other hand, I think we are going to come to grips with that later on If one therefore restricts oneself to the empirical analysis of the ballistocardiogram, much along the lines of the four grades which we classified, it offers a simple

way to separate the various abnormalities that accrue in the ballistocardiograms.

The Grade I, with the respiratory variations, increasing and decreasing, has pertinency in angina pectoris. I think that if I were rewriting the chapter in the monograph, I would now draw its final line in angina pectoris. On the other hand, I do think it stands up to date.

The Grades II, III and IV more or less speak for themselves, and I think they are of significance, and, clinically, I am finding them to be so.

CHAIRMAN STARR Here are a couple of questions from the audience. I am going to take up one that I am going to answer myself This question is, "What effect does peripheral resistance have on the ballistocardiogram?" I studied that very carefully in the cadaver preparations The question being asked is, "What effect does it have on the amplitude of the I-J distance as a measure of the heart's force?" and the answer is, I remember that I was dreadfully perplexed to find I had a great deal of difficulty finding that it had any effect at all. Eventually, by throwing in a great deal of data and using very high hypertension, I did manage to get a little significant effect. But the answer is that for clinical purposes, the effect of things like the height of blood pressure seems to be negligible, and I take it that the height of blood pressure is a measure of peripheral resistance

The next question from the audience is, "Will somebody compare the Arbeit type of ballistocardiogram with the other types?" I myself have never used the Arbeit type, though I am well aware of it. Would any member of the panel like to try that?

DR. SAMUEL A. TALBOT: I have had the pleasure today of spending an hour or so with Dr. Arbeit, and I am fairly fresh on this point

I think that the outstanding feature of this method of recording is the emphasis that it places upon the high frequency components of the ballistocardiogram. He has, of course, met the danger of ambient vibrations, and of muscular tremor, by cut-

ting off at about 20 cycles, but up to 20 cycles, he has a great deal of information in his records

In looking at the records taken at the upper end of the spectrum, which appears mainly in the acceleration ballistocardiograph, one finds the typical breaking up of the detail, the spiking and fractionation of the peaks, that also appears in Dr. Smith's records If you will remember back to the case that Dr. Baker just showed, in which the patient took a bottle of Coca-Cola on top of bicarbonate, the main feature of the resulting record was tremendous slurring and breakup of the peaks, and all kinds of high frequency detail that could not be detected upon a displacement record became evident. This kind of high frequency detail is what is mainly emphasized by the acceleration record, either by the Arbeit system or by Smith's

CHAIRMAN STARR: Here is a question addressed to Dr. Mandelbaum. The question is, "If your 70-year-old woman were asymptomatic, would you have stopped her from working if her BCG had not improved on exercise?"

DR. MANDELBAUM I would like merely to repeat what other members of the panel have stated Where the ballistocardiograph gives us results that complement our clinical impression, then it permits us to be in a position to be able to be more sure of ourselves If the clinical examination of this woman and the findings, as indicated in the basal ballistocardiograph, did not improve after exercise, I would not be inclined to stop her from her activity, because the ultimate diagnosis that supersedes everything else is the clinical diagnosis.

CHAIRMAN STARR: It appears that the time has come to close. Therefore, I am going to close by thanking the panel very much for helping me in getting our message across

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